The Journal of Thoracic Surgery

VOL. 26 DECEMBER, 1953 NO. 6

Original Communications

THE EXPERIMENTAL CLOSURE OF ATRIAL SEPTAL DEFECTS

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IT WAS the object of this study to explore various methods for surgical closure of atrial septal defects and, if possible, to develop a method applicable to human patients. It was our original feeling that the ideal procedure for use in patients would be one which would be applicable to all types and sizes of atrial septal defects, which would give fairly certain assurance of complete closure of the defect, preferably with autogenous tissue, and which could be accomplished in a dry field without loss of blood, without interference with cardiac function, without the necessity of using anticoagulants, and without the hazard of air embolism or intracardiac thrombosis.

Healthy mongrel dogs were used. A large atrial septal defect made through the opened right atrium during momentary occlusion of venous inflow was created in each animal either immediately before the closure or at some time previous to it.[1] Following induction with intravenously administered pentobarbital sodium intratracheal ether-oxygen anesthesia was employed, using an intermittent positive pressure apparatus. The heart was exposed through a long right intercostal incision. Penicillin was given postoperatively for the first week. No anticoagulants were used.

As compared with the situation which one might expect to encounter in human patients, the attempts at surgical closure of atrial septal defects in experimental animals were performed under certain handicaps. If the defect were made some time beforehand, application of the graft was facilitated because the margins of the defect were firm and took sutures well. The pleural

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Aided by a grant from the James Whitcomb Riley Memorial Association and a contract between the Office of Naval Research, the United States Navy, and Indiana University.

Read at the Thirty-third Annual Meeting of The American Association for Thoracic Surgery, San Francisco, Calif., March 27, 28, and 30, 1953.

and pericardial adhesions from the first operation, however, added to the difficulty of exposure and the previous incision through the atrial wall was particularly troublesome when the method of closure necessitated insertion of a pericardial pocket in the same area. Creation of the defect immediately before its closure eliminated the latter obstacles but the fresh pliable margins of the defect made the suturing of the graft in place somewhat more difficult than would have been the case with a defect of long duration. As a consequence, both freshly created and older defects were utilized, the former particularly in those experiments requiring the fixation of a pericardial pocket to the edges of an incision in the atrial wall.

ONLAY GRAFT THROUGH OPEN RIGHT ATRIAL CHAMBER

This group of experiments was concerned with the direct application of an autogenous graft to the septal defect through the opened right atrium during temporary occlusion of the inferior and superior venae cavae and the azygos vein.

Direct Circular Suture of Graft to Margins of Defect.—Preliminary observations revealed two facts of significance. In the first place, in contrast to the situation with the intact normal heart, the period of safe occlusion of venous inflow into the right side of the heart is markedly shortened if the right atrium is opened at the same time and a graft sutured to the margins of the defect. Templeton and Gibbon[2] found that systemic venous return to the intact normal dog's heart could be blocked for periods up to nine minutes with survival of the animal. In other experiments dealing with the production of atrial septal defects,[1] we learned that the period of venous occlusion had to be shortened considerably in order to assure survival. Though the safe upper limit was not determined statistically, we cut the period of occlusion to around one minute or less and eliminated fatalities. Similarly, in our early efforts with the grafting of septal defects it was apparent that only a brief interval of obstruction of venous inflow was compatible with survival. In the second place, it soon became evident that unsupported grafts of pericardium or of auricular appendage could not easily be sutured in place during short periods of occlusion. Suturing was time consuming even though the graft was kept flat and taut by traction on the sutures placed equidistantly along its margins. The traction sutures and the clamps attached to them got in the way and reduced the speed of suture.

We therefore resorted to means of splinting the graft. The grafts were made either of auricular appendage or of pericardium. When the appendage was used it was excised competely after clamping its base. The pectinate muscles which joined the two walls were divided and a circular ring of stainless steel wire was inserted into the cavity. The open margins of the appendage at the base were then closed with a continuous inverting suture of 00000 Deknatel (Fig. 1, *a* and *b*).

Pericardial grafts were made in similar fasion. A segment of pericardium of suitable size, rectangular in shape and roughly twice as long as wide, was excised. The pericardium was then folded on itself so as to make a square of double thickness with the external surface of the pericardium exposed.

Two of the three open sides were closed with continuous sutures. The pocket was everted so as to have the smooth inner surface outside. The wire ring was inserted and the remaining open side closed with an inverting suture (Fig. 1, *c*). Thus both types of graft were covered with smooth uninjured mesothelium. By clamping the ring at one point through the overlying tissue, the disclike graft could be held perpendicular to the long axis of the clamp. A suture was placed opposite the clamp and the ends left long. As soon as the atrium was opened, the graft was placed over the defect and the previously placed suture was passed through the margin of the defect and

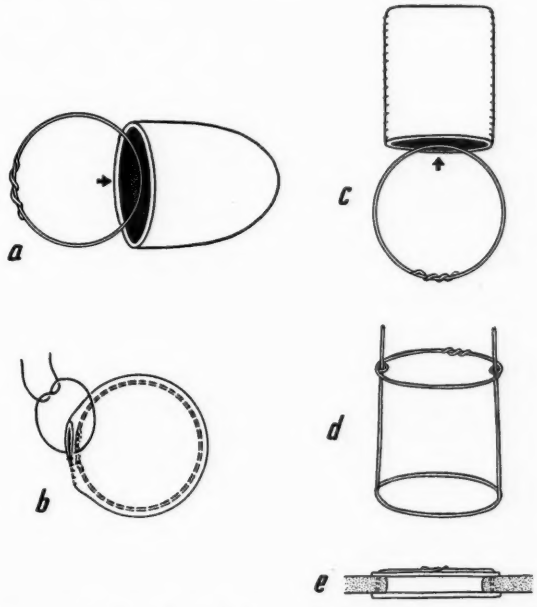

Fig. 1.—Diagrammatic illustration of method of constructing wire ring-splinted grafts of atrial appendage (*a* and *b*) and pericardium (*c*) and method of fixing double onlay graft with wire sutures (*d* and *e*).

tied. The suture was then continued around the periphery, fixing the margins of the graft to the edge of the defect. After a brief period the clamp was removed, the suture brought out through the cardiotomy opening which was closed temporarily with a Satinsky or an angulated Potts or Glover clamp, the vena caval obstruction released, and the lungs hyperventilated with oxygen. A complete circular suture could generally be completed during three short periods of vena caval occlusion. The results of such direct continuous suture are exemplified by the following experiments:

Dog 93.—A large septal defect was made during a forty-second period of venous obstruction. A splinted graft of auricular appendage was sutured in place with a continuous suture of fine steel wire during three periods of occlusion of venous inflow of 95, 117, and 158 seconds' duration. Heart action was never properly established after the last period and

ceased shortly afterward. Post-mortem examination revealed the defect to be tightly closed. There was no obstruction to the ostia of the venae cavae, nor were there any thrombi upon the graft or within the heart.

Dog 94.—A large septal defect was made. In three sessions a splinted circular graft of auricular appendage was sutured in place with a continuous wire suture. The three periods of vena caval occlusion averaged eighty-eight seconds, the total period of occlusion being four minutes and twenty-three seconds. The animal was sacrificed two days later. The graft was securely sutured to the margins of the defect closing it tightly, and no thrombi were present. The wire ring lay within the graft.

Dog 97.—A large septal defect was made. This was followed by suturing in place a splinted pericardial graft with a continuous wire suture during three periods of obstruction of systemic venous inflow of approximately the same duration as in Dog 94. The animal was sacrificed nine months later. The graft was well healed to the septum and the defect was completely closed. A portion of the wire ring projected into the right atrium caudally, lying alongside the posterior portion of the inferior vena caval ostium. There was no obstruction and no thrombi were present.

Fixation of Graft by Sutures Passed Through Septum and Out Through Atrial Wall.—In order to shorten the period required for fixing the graft to the septum, a number of modifications were tried. In some animals, five silk sutures were placed through the rim of the splinted graft, equidistant from one another, and tied. Both ends of one suture were left long. One end of each of the other sutures was cut short while the end to which the needle was attached was left long. As soon as the right atrium was opened, the first suture was passed through the posterior margin of the defect and tied. The other four were then passed through the septum and out through the atrial wall. One suture went through the posterior caudal lip of the defect and out of the atrium under the inferior vena cava. Another passed through the posterior caudal lip of the defect and out of the atrium under the inferior vena cava. Another passed through the posterior cephalad portion of the margin of the defect and emerged underneath the junction of the superior vena cava and the auricular appendage. The other two sutures were directed through the anterior rim of the defect and out through the interatrial groove. A clamp was placed across each suture at the point where it passed out of the atrium in order to keep it taut so the graft would be held in proper position. After closing the cardiotomy opening, each suture was secured by tying it to a fine silk suture passed through the adjacent auricular wall. The results of these efforts are illustrated by the following experiences:

Dog 115.—Closure with an auricular appendage graft was attempted by the method outlined fifteen days after creation of a large defect. The procedure required obstruction to venous inflow for seventy-five seconds. Six days later the dog died of pneumonia. The graft was in place and the defect was closed in large part. The posterior caudal suture had pulled out of the septal margin and a tiny defect had resulted. No thrombus was present.

Dog 117.—Thirty days after the production of a large septal defect it was repaired in the same manner as that used in Dog 115. Forty-six days later the animal was sacrificed. The graft was securely healed to the margins of the defect, closing it completely. There was no thrombus and the graft was covered with smooth glistening endothelium. A small portion of the wire had eroded into the left atrial cavity along the caudal portion of the graft.

Other Modifications.—Another method utilized a double pericardial graft, each of which was stretched over a wire ring. To one of the rings were fixed two wire sutures, opposite one another (Fig. 1, *d* and *e*). The other ring had two small loops opposite one another. The first graft was introduced into the atrial chamber and pushed through the defect into the left atrium. The two wire sutures were directed through the margins of the defect and out into the right atrium. They were then passed through the tiny wire loops of the other graft which was pushed down against the defect. The two sutures were quickly tied over this graft so as to hold it snugly against the other with the rim of the defect interposed between the peripheral portions of each. The excess wire was cut away and the cardiotomy closed. The results are illustrated by the following example:

Fig. 2.—Photograph of septum in Dog 191 three months after repair by double onlay wire splinted pericardial graft. The erosion of the wire rings into the atrial cavity is seen.

Dog 191.—A large septal defect was created and repaired at the same operation. The animal was sacrificed three months later. The graft was smooth and firmly united with the septum. The original defect was so nearly completely closed that there appeared to be no functioning defect though actually a tiny opening remained. The wire rings were not visible from the left atrial side. Through the right atrium one could see a small segment of the deeper ring covered with endothelium (Fig. 2). About 40 per cent of the larger ring of the second graft had eroded through. The wire suture over the graft could be seen under a thin endothelial cover. There was no thrombus and no obstruction to the ostia of venae cavae or coronary sinus.

Other measures were employed with less success. For example, a splint was constructed with two cross bars passing at right angles across the ring.

The four ends of the cross bars were twisted about the ring and cut about 4 or 5 mm. long so as to leave four sharp spikes projecting perpendicularly to the plane of the splint. The splint was covered with a graft. The mid-portion where the cross bars intersected was grasped with a clamp. The graft was thrust through the defect into the left atrium and an attempt was made to pull it upward against the septum so that the four spikes would perforate the septal margins. This maneuver was found to be awkward and generally unsatisfactory. Some of the spikes bent and did not perforate the septum. Sometimes one or more would come through properly while others would slip into the defect. Furthermore it was usually not possible to bend them so as to engage the graft firmly to the rim of the defect. Various modifications were tried but none proved satisfactory.

Comment.—It was apparent that the direct application of onlay grafts to atrial septal defects by direct suture during periods of obstruction of venous inflow were attended by sufficient hazards to make its use in human patients unjustified.

The safety of the operation could be increased by various modifications which enabled the surgeon to fix the graft in place during a single brief period of venous obstruction. For those who may attempt such direct repairs through the open atrial chamber, either under conditions of hypothermia or during exclusion of the heart with the use of an extracorporeal pump-oxygenator apparatus, certain of our experiences may prove helpful. First, grafts made either of auricular appendage or of pericardium, so constructed that their external surfaces presented an uninjured smooth mesothelial surface, appeared to be entirely satisfactory in the dog. They united well with the septum and no gross thrombi developed upon them. As will be developed subsequently, however, it is doubtful that large free grafts of the thicker human pericardium or atrial appendage will prove satisfactory and it may be preferable to utilize inert plastic discs. Second, the suturing of free autogenous grafts in place was greatly facilitated by splinting them. Though the circular wire splints used never became completely dislodged and, during the period of observation, never appeared to cause difficulty, they tended in time to erode partially through the graft and to project into the atrial cavity. Perhaps the same end could be accomplished by some absorbable splint such as a fibrin ring or disc. It would probably be preferable to utilize inert plastic discs instead of autogenous tissue. Third, the free circular grafts were more reliably fixed so as to accomplish complete closure when they were united to the margins of the defect by direct continuous suture. Multiple interrupted sutures would seemingly also be satisfactory. The use of multiple sutures tied to the edge of the graft and thrust through the septum and out through the atrial wall reduced the speed of application of the graft and was reasonably reliable though it was more often followed by persistence of small defects. If this method were combined with the use of a few direct sutures of the graft to the margin of the defect the percentage of complete closures would undoubtedly be increased.

INSERTION OF GRAFT THROUGH A RUBBER-CAPPED CYLINDRIC GLASS WINDOW

Glass cylinders about 2 cm. long and of varying diameter were constructed. A ridge was fashioned near either end in order to keep an encircling ligature from slipping off. A piece of rubber dam was secured over one end with a ligature (Fig. 3, *a*). A purse-string suture was placed about the mid-portion

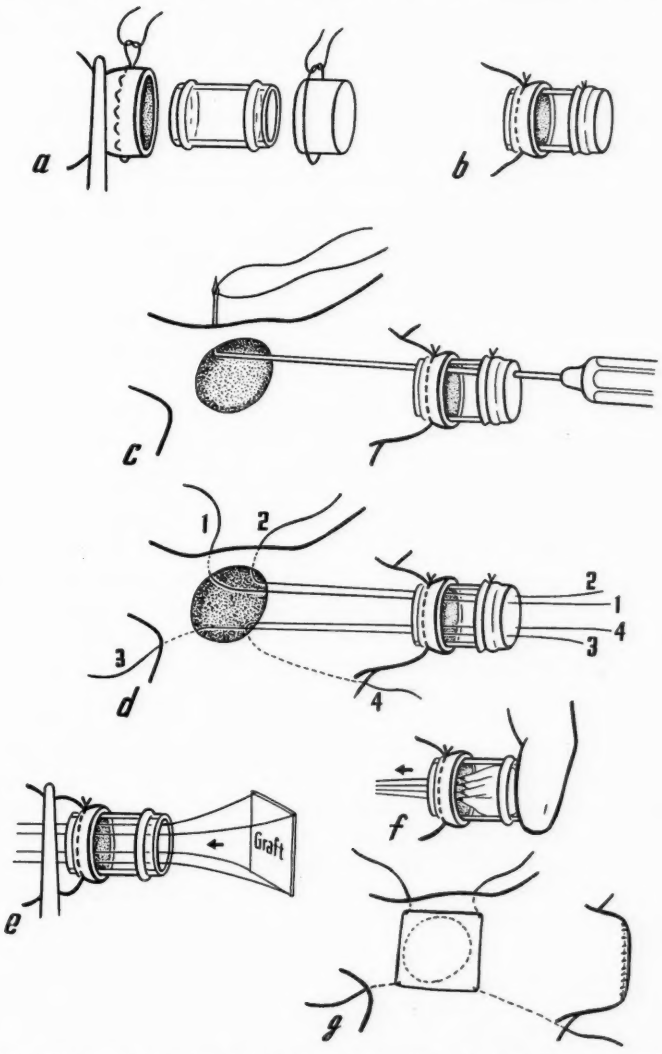

Fig. 3.—Drawings illustrating technique of insertion of graft through rubber-capped glass cylinder. In *a* and *b* the covering of the cylinder with a rubber cap and its insertion into the atrial appendage are shown. In *c* and *d* are illustrated the placement of sutures through the septal rim and atrial wall. *e* and *f* show the pulling of the graft into the atrial chamber without loss of blood; in *g* the graft is in place.

of the right auricular appendage. The appendage was clamped at its base, the tip amputated, the pectinate muscles divided, and the cylinder fixed in place by drawing the suture taut about its base (Fig. 3, *b*). The clamp was released and blood allowed to flow up into the cylinder. A needle was introduced into the cylinder through the rubber diaphragm and a small amount of heparin solution injected. Air was usually not present in discernible quantity. If present, it would rise to the top of the cylinder and could be withdrawn by aspiration into a syringe.

A modified small aneurysmal needle with the end sharpened and made into a right angle curve was passed through the rubber cap and cylinder into the right atrial chamber (Fig. 3, *c*).

Ordinarily it was easy to locate the defect and to pass the needle through it into the left atrium. By bringing the end of the needle up against the left atrial wall just beyond the septum one could see it push the wall out, feel it, and be certain that it had passed through the defect into the left atrial chamber. It was then passed through the left atrial wall anteriorly near the cephalad margin of the defect. A fine wire or silk suture was threaded onto the needle. One end was held while the needle was withdrawn through the defect, the right atrium and the cylinder.

In succession three other sutures were placed (Fig. 3, *d*), one emerging from the left atrium near the anterior caudal portion of the defect, one through the posterior caudal portion of the defect and emerging from the posterior aspect of the atrial wall near the inferior vena cava, and one through the posterior cephalad portion of the septum and passing out of the atrial wall near the posterior surface of the atrium between superior vena cava and appendage. The cylinder was elevated, the base of the appendage again clamped, the rubber dam removed and stripped off the sutures, the cylinder irrigated with saline solution and the four sutures fixed to the four corners of the graft (Fig. 3, *e*). By drawing up on the various sutures which had been passed through the atrial wall while the clamp was loosened slightly the graft could be pulled down into the glass chamber. The top of the cylinder was then covered with a finger, the clamp released and the graft drawn into the atrium (Fig. 3, *f*) and snugly up against the septal defect. The base of the appendage was then clamped, the glass cylinder removed, and the opening subsequently closed with silk sutures (Fig. 3, *g*). Each of the sutures attached to the graft was fixed to the atrial wall at its point of emergence by tying it to a fine silk suture placed in the atrial wall. The graft could thus be inserted without loss of blood and in no case was clotting a problem, nor air embolism.

This principle was employed with varying success in nine animals. In one the graft was a wire ring-splinted pericardial graft, in two a segment of inverted external jugular vein, in six a pericardial graft constructed in the manner previously described but without a wire splint. Wire sutures were used in seven animals, silk sutures in two. In one case, a modification of the method of placement of the glass cylinder was used, the cylinder being in-

serted into the azygos vein as it entered the superior vena cava. In the remainder the cylinder was inserted through the base of the appendage. The attempts at closure were carried out from sixteen to fifty-three days after creation of the septal defect.

The chief obstacles encountered were the occasional crossing of sutures as they were inserted and the difficulty of accurate placement of the two posterior sutures. The results varied. In most of the animals the original septal defect was closed in its anterior portion but the posterior third of the defect remained open. In only one case was complete closure secured. The sutures caused no intracardiac thrombosis and were often not visible at the time of final examination, being buried in the septum. The results obtained are illustrated by the following experiences:

Dog 196.—An atrial septal defect approximately 1.7 cm. in diameter was made. Seven weeks later using a pericardial graft and wire sutures closure was attempted. Before closing the opening in the base of the appendage a finger was introduced into the right atrium and the area of grafting palpated. The posterior sutures had not been placed accurately and the posterior part of the defect was not completely closed. The animal was sacrificed six and one-half months later. The anterior portion of the defect was nearly closed but approximately one-fourth of the original defect was open in its posterior part. The graft was nicely united to the septal wall and was covered with glistening endothelium. The sutures were not visible on the interior of the heart and there were no thrombi.

Dog 199.—Two and one-half weeks after creation of an atrial septal defect approximately 1.8 cm. in diameter, repair was carried out in the manner employed in Dog 196. The heart was examined two weeks later. The defect was closed completely. The graft appeared well united with the septum. There were no intracardiac thrombi.

Comment.—The principle employed has certain virtues. The method permitted the insertion of a graft into the interior of the heart without interfering with cardiac function, without loss of blood, and without apparent hazard of intracardiac thrombosis or air embolism. Use of the base of the appendage for insertion of the cylinder, however, fixed the cylinder some distance from the defect and, in spite of the fact that it could be brought nearer the defect by virtue of the mobility of the atrial wall, made accurate placement of sutures somewhat difficult. It was apparent that the method might be modified to advantage. If the larger glass or plastic cylinder was introduced through the atrial wall near the site of the defect it would appear evident that sutures could be placed with greater accuracy, crossing of sutures avoided, and complete closure more often achieved. Furthermore, a finger introduced through the atrial appendage would facilitate placement of sutures. It is planned to investigate this matter in the near future and to use plastic rather than viable grafts.

CLOSURE OF DEFECT BY USE OF A PERICARDIAL POCKET AFFIXED TO AN INCISION IN THE ATRIAL WALL

The operative technique finally evolved is as follows[3]: A large segment of the presenting pericardium is excised after removing any fatty tissue presenting on its external surface. It is trimmed to a roughly square shape ap-

proximately 6.5 cm. in diameter. Care is taken to keep the graft moistened with physiologic saline solution and to avoid injury to the smooth cardiac surface. The square is folded on itself with the external surface outside and each of the two shorter open sides of the resultant rectangular graft are closed with a fine silk purse-string suture (Fig. 4). There results a pocket open at one end.

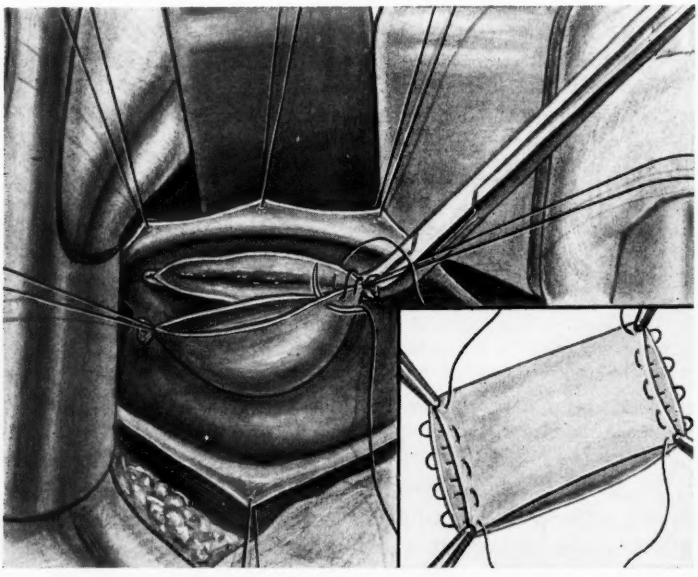

Fig. 4.—Drawing illustrating technique of construction of pericardial pocket and its fixation to an incision in the atrial wall.

Traction sutures are placed in the atrial wall roughly parallel to the interatrial groove, the lowermost suture being in the anterior wall of the proximal end of the inferior vena cava, the uppermost near the end of the superior vena cava, or, in case the heart is small, more cephalad near the base of the atrial appendage. During momentary occlusion of the systemic venous inflow, an incision is made and a defect of the atrial septum roughly 2 cm. in diameter is created. The atrial incision is closed with a modified angulated Glover clamp and the venous obstruction released. The open edges of the pericardial pocket are then sutured to the edges of the incision using a continuous 00000 silk suture interrupted in at least one place (Fig. 4). On release of the clamp the pericardial pocket fills with blood and bulges out. It is now invaginated into the atrial cavity, its smooth and uninjured cardiac surface presenting on the interior of the heart. Its posterior wall is pushed against the septum and the defect can be accurately palpated and outlined. The tip of the left index, and sometimes that of the middle finger, is kept in the pocket and serves both to retract the anterior and to push the posterior

wall snugly against the septum. With practice the posterior wall can be kept in proper position so as to outline the margin of the defect and the excess of the pocket can be kept from ballooning out into the field and obscuring vision. A 0000 catgut suture is then passed through the pericardium and the anterior lip of the margin of the defect. It is tied and continued as a locking suture around the rim of the septal defect (Fig. 5). At the completion of the closure it is tied to the other end of the suture. If desired it can be supplemented by a few interrupted sutures. By its purse-string effect, it not only seals off the defect with the pericardial graft but also reduces the cross-sectional area of the defect. If at the completion of the procedure there is no excess of pericardium, the lungs are inflated and the chest wall closed. In rare instances there is an excess of pericardium. In such cases a portion of the pocket bulges out of the atrium. The protruding pericardium is then clamped, amputated, and the cut margins approximated with a fine silk suture.

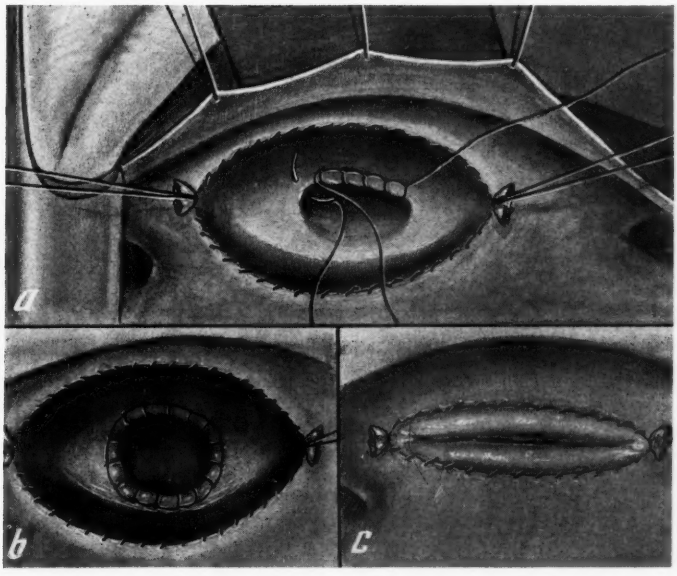

Fig. 5.—Illustration of the method of suturing the posterior wall of pericardium to rim of the septal defect. *a*, The suture is started. *b*, The suture is completed. *c*, Appearance of atrial wall at completion of operation.

In actual practice the defects made were often so large as to leave no rim of septum anteriorly. In such cases the sutures were passed from pericardium out through the left atrial wall immediately adjacent to the interatrial groove and back again through the pericardium. Often, too, there was no palpable rim at the inferior aspect of the defect. In such cases sutures were passed out through the left atrial wall alongside the posterior aspect of the proximal end of the inferior cava and back through the pericardium.

During our early experiences various methods of suture were tried, and various sizes and shapes of pericardial pocket were tested. In some instances

the pocket was made in a rectangular fashion by closing without purse-stringing the two shorter ends of the overfolded pericardium. In some a triangular pericardial pocket was constructed.

In nine dogs the posterior wall of the pocket was fixed to the rim of the defect by interrupted silk sutures, often fairly widely spaced. The hearts were examined from three to seventy-two days later. In three dogs a fairly sizable residual defect was found, in three dogs closure was complete, while in the others closure was practically complete, the residual defects ranging in diameter up to 4 mm. It is important to remember that when a defect 2 cm. in diameter is reduced to one 4 mm. in diameter the surface area of the opening has been reduced by over 96 per cent and when reduced to one 2 mm. in diameter by 99 per cent.

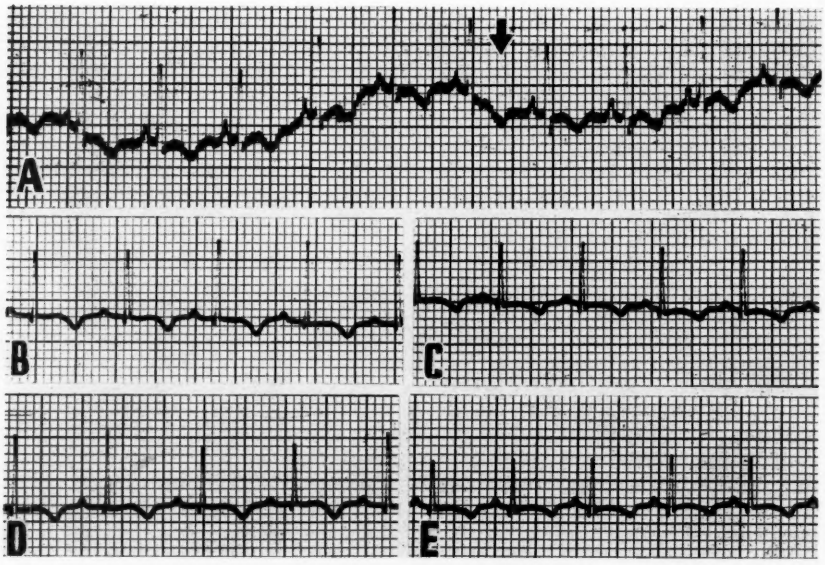

Fig. 6.—Electrocardiographic tracings during operative repair of atrial septal defect by the invaginated pericardial pocket technique. *A*, Lead II tracing during clamping of atrial wall (arrow) after creating septal defect. *B* and *C*, Lead II tracings and *D* and *E*, Lead III tracings before and immediately after repair of defect.

In four animals the posterior wall of the pocket was united with the margin of the defect with Michele clips applied with a specially designed instrument. One defect was only half closed; in one dog there was a residual opening 4 mm. in diameter, and in two dogs closure was complete. An occasional clip was seen to have eroded partially into the atrial cavity though none was dislodged.

In two dogs a continous fine silk suture was used. In one animal examined three months later there was a tiny opening 2 to 3 mm. in diameter. Closure was complete in the other which was examined four months after repair.

Small silk granulomas were observed between the adherent anterior and posterior layers of pericardial pocket.

For this reason it was decided to employ a catgut suture. These efforts are considered in two groups. The first were made with pericardial pockets of varying size and shape and before we were aware of the extreme importance of utilizing an incision in the atrial wall of adequate length. Closure in this group of nine animals, examined from two to fifty-three days after repair, was either complete or nearly so, residual openings varying in size from 1 to 3 mm. in diameter. The second group of nine dogs was operated upon after the previously described technique had been fully developed. One heart was examined five days, one twenty-nine, and one thirty-seven days after operation. The others were studied from six to seven weeks after repair. Closure was complete in seven while in two a pin-point opening 1 mm. in diameter could be found. Since the results were consistent the series was not enlarged.

Fig. 7.—Photograph of atrial septum of Dog 325, six and one-half weeks after repair of defect as seen from left atrial side. The grafted area is hardly distinguishable from the remainder of the septum.

The operation was tolerated well. In the entire group treated by the inverted pericardial pocket technique there were no immediate operative deaths. In the early experiments there were some deaths later from empyema and pneumonia. Except for these animals, one which was badly injured in a fight with another dog and one which was sacrificed because of cerebral dam-

age suffered at the time of operation while an empty oxygen tank was tardily being replaced, all the animals quickly recovered from the procedure and remained in excellent health. No significant electrocardiographic alterations were noted after making the atrial septal defect, after placing the clamp on the atrial wall, or after repairing the defect (Fig. 6).

Fig. 8.—Photograph showing repaired septum in Dog 338, five and one-half weeks after repair as seen from right atrial side. The ostia of the venae cavae and coronary sinus are unobstructed. It can be seen that the original defect was practically complete extending from the inferior to the superior vena caval ostium and down to the floor of the atrium.

The two layers of the pericardial pocket quickly became sealed together. The graft became smoothly fused with the septum. When examined some time after repair the graft was covered with smooth endothelium. Intracardiac thrombi were not observed. From the left atrial side the grafted area was hardly distinguishable from the remainder of the septum (Fig. 7). In no case was there encroachment upon the pulmonary veins. From the right atrial side the thickened area of the pericardial graft was more readily identified. It was covered with smooth endothelium and did not encroach upon the ostia of the venae cavae or coronary sinus (Fig. 8). Microscopically the fibrous tissue of the graft was firmly united with the muscular tissue of the septum and was covered with smooth endothelium (Fig. 9).

Comment.—This method seemed highly satisfactory and eminently suitable for use in human beings. Actual experience[20] has shown that the procedure is withstood well by patients and that the defect can be securely closed at the time of operation. It has become evident, however, that the pericardium placed over a large defect may disintegrate completely, presumably from failure of sufficiently rapid vascularization of the free transplant. It might be supposed that the method could be successfully modified by employ-

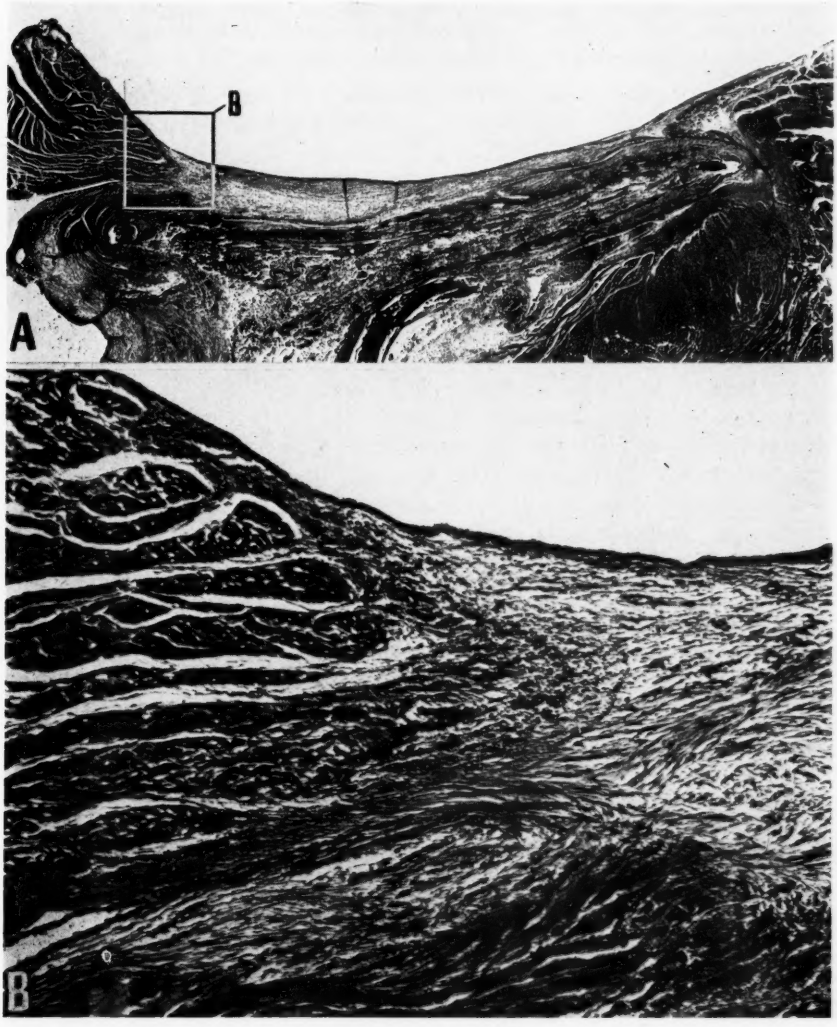

Fig. 9.—Photomicrograph of specimen from Dog 349 showing firm union of pericardial graft and septal wall six weeks after operation. The graft is covered with endothelium.

ing a pocket of inert plastic material and suturing the incised atrial wall carefully after completing closure of the defect. Such experiments have been initiated. They have thus far shown that the modification is technically satisfactory and is withstood well by the animals. A second modification is also being studied and is similarly technically feasible and well tolerated. This involves direct suture of a plastic disc to the rim of the defect through the invaginated pericardial pocket. Both methods are to be carefully evaluated. The first seemingly is more promising for human application since it eliminates the possibility of intracardiac disintegration of pericardial tissue.

DISCUSSION

Before the studies described were begun a number of reports had appeared in the literature describing surgical efforts to close atrial septal defects produced experimentally in animals or occurring spontaneously in human patients. In 1947 Cohn[4] made small atrial septal defects by passing a clamp into the left atrium through the appendage, thrusting it blindly through the septum, and opening the blades of the instrument. The auricular wall was subsequently invaginated into the right atrial cavity, fixed to the margin of the defect by one or two sutures, the tip of the invaginated portion severed by a wire snare, and the opening in the atrial wall closed by drawing taut a previously placed purse-string suture.

In 1948 Murray[5] reported that he had closed atrial septal defects in about thirty dogs by passing sutures in the line of the interauricular septum beginning at the site of the transverse sinus behind the aorta and coming out posteriorly between the superior vena cava and the right pulmonary veins in such a way that when two, three, or four such sutures were pulled taut the anterior and posterior atrial walls were compressed together and the defect occluded. The size of the defects was not mentioned. He described one patient who had improved following this procedure.

In 1949 Dodrill[6] recorded his experiences with the use in dogs of a specially designed clamp which permitted compression of the right and left atrial walls against the septum. By this means he was able to excise segments of the septum and to repair them by direct suture approximation of the cut edges or by suturing the rim of the defect to the left atrial wall.

Santy and his associates[7] described, in 1950, the plugging of a small septal defect in a patient by inverting the tip of the left auricular appendage through it. In the same year Swan and his associates[8] reported efforts to close defects in dogs made by direct excision of a portion of the septum through the opened right atrium. They inverted both the right and left atrial appendages by tightening a silk suture which had been passed from the right to the left appendage through the atrial chambers and the defect and onto which were threaded two circular buttons of stiff polyethylene, each of which was then invaginated into one of the appendages. This operation was carried out upon ten dogs, eight of which were examined after they died or were sacrificed. The defects were rather small, only one of them being of greater diameter

than 1 cm. At times thrombosis occurred and occasionally this process completed the closure of a defect which had been incompletely closed by the operative procedure.

Since our own experiments were begun a number of additional reports have been recorded. In 1951 Martin and Essex[9] described the use in eight dogs of a small polyethylene disc, either bare or covered with an inverted segment of azygos vein. The defects were created immediately before closure was attempted and were made by blind incision of the septum with a knife inserted through the auricular appendage. The discs measured either 1 by 1, or 1 by 2 cm. in size. They were introduced through a small atrial incision after three attached sutures had been passed blindly through the septum and out of the atrial wall. The defects were very small and the authors reported considerable variation in the accuracy of placement of the graft. They abandoned efforts to close defects through the opened right atrium during temporary vena caval occlusion because of the prohibitively high mortality. In the same year Hufnagel and Gillespie[10] reported the successful closure of defects by means of a plastic prosthetic device consisting of two discs which could be secured together, one on either side of the septum. In 1951, Dennis and his associates[11] also recorded their fatal effort to close a septal defect through the opened atrium in a patient in whom a pump-oxygenator was used to replace heart and lung function.

The following year Kiriluk, Hoag, and Merindino[12] described the creation through the opened right atrium of large septal defects which tended to remain open during long periods of observation. They attempted closure by three methods: the suture of an infolded segment of auricular wall into the defect, the direct closure by suture of the defect through the opened atrium, and the nonsuture plugging of the defect by a specially constructed pericardial graft introduced through a biauricular approach. The closure was complete in four of six dogs treated by the first method, five of six treated by the second, and three of six treated by the third.

After our studies were completed additional reports appeared in the literature. Cookson, Neptune, and Bailey[13] recorded experiences with the use of hypothermia in human cardiac surgery. They included a fatal case in which repair of an atrial septal defect was attempted through the opened right atrium. The patient died of ventricular fibrillation. The authors concluded that open operations for closure of atrial septal defects were contraindicated because of the hazard of air embolism.

Subsequently Lewis and Taufic[14] reported the case of a 5-year-old girl successfully treated during a period of hypothermia by direct closure of a septal defect through the opened atrium. They operated upon twenty-six hypothermic dogs for closure of experimentally produced atrial septal defects. One defect was found to have closed spontaneously. Seventeen dogs survived opening the atrium and repairing the defect. There were eight deaths, four from ventricular fibrillation. In two of the latter there was obvious coronary air embolism. Seven additional animals had ventricular fibrillation but recovered. One death occurred in the last ten closures.

Swan[15] has recently described his clinical experience with the use of the method of biauricular invagination previously developed in experimental animals. One patient died and two survived the operation. In both the surviving patients postoperative cardiac catheterization studies were disappointing since a significant left-to-right atrial shunt was still present. A modified procedure was utilized in two patients, both of whom lived and in both of whom the defect was estimated to have been about 80 per cent closed. A finger introduced through the right atrial appendage was used to guide two sutures from the left auricular appendage through opposing edges of the defect and out through the incision in the right appendage. A plastic button threaded onto these sutures was pushed into the right atrium after momentarily withdrawing the finger. A second button was threaded onto the sutures outside the left appendage and thus the left appendage and the button in the right atrium could be brought together against the septal defect.

Bailey and his associates[13] had found Swan's original method unsatisfactory. One patient went steadily downhill after an attempt at closure and was reoperated upon for removal of the buttons. He died two days later. Another who underwent mitral commissurotomy as well as closure of an atrial septal defect did not survive. A third died thirteen days after operation. Post-mortem examination revealed the defect to have been only partially closed. No healing had taken place between the invaginated appendages and the rim of the septal defect. Two patients survived operation but had persistent evidence of atrial shunt. Because of these experiences another procedure was proposed. Using for guidance a finger introduced through the right atrial appendage the right atrial wall was sutured to the rim of the defect. Two patients were so treated with success.[13, 16]

Gross and his associates[17-19] have recently described their experiences with a method of repair involving the use of a large rubber well which is sutured to the atrium. The auricular wall is incised and blood flows up into the well. Intracardiac manipulations are carried out by palpation through the pool of blood. Clotting is prevented by intermittent addition of heparin to the blood in the well. Using this procedure closure was carried out in sixty-eight dogs by suture application of auricular appendage, vein or pericardial grafts, by onlay grafts of plastic material sutured to the defect, by nonsuture application of Hufnagel-Gillespie plastic buttons and by direct suture closure of the defect. The results were highly satisfactory, the closure being complete in sixty animals and residual defects being 4 mm. in diameter or less in the remainder. Seven patients were operated upon. In three, plastic buttons were used. All died. In three, nylon or polyethylene was sutured to the defect. Two survived. In one patient, who did well, a small defect was closed by direct approximation of its edges, a second defect by suturing its rim to the atrial wall.

In spite of these varied and excellent contributions it would seem fair to conclude that no method thus far described is ideal.

Our studies have revealed several facts of interest. The experiments with direct application of overfolded segments of atrial appendage or of pericardium

demonstrated that such grafts are entirely suitable in the dog, unite firmly with the septum and do not cause intracardiac thrombosis. It is doubtful, however, whether such free autogenous grafts will prove satisfactory for the closure of large defects in human beings. The introduction of grafts through the opened atrium during momentary occlusion of venous inflow was found compatible with survival but was considered too hazardous for use in patients.

The likelihood of survival was related to the speed of application. The period of necessary vena caval occlusion could be shortened by carrying out the process of suture in stages with several short rather than one longer period of venous obstruction and by several modifications which increased the rapidity of fixation of the graft. The experiences with the use of a rubber-capped glass chamber introduced into the base of the appendage showed this method to be safe and unattended by hazard of air embolism or intracardiac thrombosis. The position of the chamber somewhat distant from the defect resulted in some difficulty with accurate placement of sutures and suggested the likelihood that fixation of a larger chamber in the atrial wall near the defect might overcome this trouble. Such a modification is to be studied in the near future.

The technique involving the use of an invaginated pericardial pocket gave satisfactory results and after the most careful evaluation seemed entirely suitable for use in human beings. It appeared to fulfill the requirements of the ideal operation. It does not interfere with cardiac function, is accomplished in a dry field without loss of blood, uses autogenous tissue, permits direct suture of graft to the rim of the defect, entails no need for anticoagulants, and seemingly is without danger of intracardiac thrombosis or air embolism.

Experience with one patient, however, has demonstrated that the pericardial pocket, when placed over a large defect may be resorbed and literally melt away.[20] Experiments are now under way in which the same technique is being employed utilizing a pocket of inert plastic material instead of pericardium. It is hoped they will prove successful and provide a method suitable for the repair of defects in patients.

CONCLUSIONS

Experiences with several methods for the surgical repair of atrial septal defects have been described. One method in which a graft is inserted through a rubber-capped glass cylinder into the atrial chamber and fixed to the rim of the septum without blood loss appears capable of modification to overcome its present deficiencies. Another technique involving the use of a pericardial pocket invaginated into the atrial cavity seemed entirely suitable for use in human beings. Actual experiences in patients having shown, however, that the pericardial graft may melt away from the area of the defect, it is apparent that autogenous pericardium is unsuitable and it is suggested that the same general technique utilizing an inert plastic pocket may prove satisfactory.

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DISCUSSION

DR. CHARLES P. BAILEY, Philadelphia.—I have certainly enjoyed this paper, but would like to present a method which we have used on a number of human beings.

(Slide.) This method has been described as atrio-septo-pexy and has been reported in the *Annals of Internal Medicine*. The operator's ungloved left index finger is inserted through the right atrial appendage to guide the passage of the suturing needle.

(Slide.) Here you see the sutures being placed and the greatly dilated right atrial wall being invaginated and accurately sutured to the periphery of the defect.

(Slide.) That results theoretically in reduction of the great atrial chamber to a doughnut-shaped chamber, the defect being bridged by a portion of the normal right atrial wall.

(Slide.) However, sometimes with a large defect you have to use up half the doughnut and just preserve a passageway for the superior vena caval flow to the remainder of the formerly dilated atrium.

(Slide.) In cases complicated by associated mitral stenosis, the finger should be passed through the defect and either a digital or a digitally guided incisional commissurotomy should be performed. Then the defect should be closed.

(Slide.) In cases with associated anomalous pulmonary venous drainage, the defect will not be a central one but will be located close to the mouths of these veins, posteriorly. It is then possible to suture the redundant atrium to the septum in such a way that two compartments are created from the right atrium. The smaller drains the anomalous pulmonary venous flow through the defect into the left atrium; the larger one drains the two venae cavae through the tricuspid valve.

(Slide.) These are our results in defects of the septum secundum. In fifteen human beings we had two with associated mitral stenosis and five with anomalous pulmonary drainage who were treated as just described. The mortality was 13.3 per cent. Marked clinical improvement was obtained in all survivors. Eight cases have been catheterized postoperatively and six have been shown to have complete abolition of the shunt.

We have done this also in seven cases of persistent ostium primum. One of those was done only two days ago; that is a short follow up, but the patient seems to be in pretty good shape. We have lost three patients with persistent ostium primum, one because of unwise use of hypothermia, one because of postoperative anuria, and one because of the poor condition of the patient. We now know, however, that the defect can be completely closed by this technique.

It seems to us that this is a very simple surgical procedure which can be easily applied in nearly all cases with this condition.

DR. EARLE B. KAY, Cleveland.—I have employed essentially Dr. Bailey's technique in the closure of atrial septal defect in eight patients, and would like to review this experience with you. Four of the eight patients had complete absence of the atrial septum. The septal defect varied from 1.5 to 3 inches in diameter. In three of the patients the septal defect was incomplete and measured 1 to 1.5 inches in diameter. In one patient the defect was very large but fenestrated. The patients ranged from 9 to 49 years of age. Two of the patients had an associated Lutembacher syndrome.

(Slide.) The first slide demonstrates the auricle after it has been invaginated and sutured to the rim of the septal defect. In those patients in whom there is complete absence of the septum, a little ridge remains at the site of the septum that can be palpated. The suture is inserted through the wall of the enlarged right auricle, through this rim of residual septum and then out through the auricular wall in a manner similar to a purse-string suture, or individual sutures from an anteroposterior direction may be employed. The two patients with an associated mitral stenosis had the commissures opened by finger traction inserted through the right auricle and septal defect.

(Slide.) The next slide demonstrates the largest giant auricle encountered. The right auricle measured over 4 inches in diameter and the septal defect measured 3 inches in diameter. The auricular wall was largely replaced by scar tissue so that their technical difficulties when associated with a rheumatic myocarditis were more serious.

(Slide.) This demonstrates the postoperative appearance of the right auricle after it has been invaginated and used to close the septal defect in this patient.

(Slide.) This slide demonstrates a postoperative angiocardiogram. The doughnut configuration of the right auricular chamber after invagination of the auricular wall is apparent.

In this series of eight patients there were three deaths. One of these was the patient previously demonstrated who had a Lutembacher syndrome associated with a giant right auricle. This patient, as well as the other two who died, had evidence of arterial desaturation.

The problem of the management of the patient with atrial septal defects who develops evidence of arterial desaturation deserves special emphasis. As a result of the increased pulmonary blood flow, increased pulmonary arterial tension is present. This may manifest itself in two ways. The increased pressure causes atheromatosis changes in the pulmonary vascular bed as demonstrated in the present slide. Often these changes are greater in the smaller pulmonary arteries and arterioles than in the capillaries. This further increases the pulmonary resistance and in turn the pulmonary arterial pressure. At this stage the increased pulmonary blood flow from the atrial septal defect may be necessary to assure proper oxygenation. Should the defect be closed, and the blood flow reduced, cyanosis may develop or increase. Furthermore, as the pulmonary pressure increases, the pressure within the right

ventricle increases and, in turn, the pressure within the right auricle may increase to the degree of causing reversal of flow, i.e., a right-to-left shunt may develop and be responsible for unoxygenated blood getting into the systemic circulation.

The two patients with evidence of arterial desaturation who died had right ventricular and pulmonary arterial pressures of 95 and 125 mm. of mercury, respectively. The arterial oxygen concentrations showed 83 and 89 volumes per cent. All three of the patients died within a period of four to five days of increasing respiratory failure and cyanosis. Autopsies in all three cases showed evidence of thrombosis of the main pulmonary arteries which was interpreted as being the result of increased pulmonary tension and stasis of blood flow.

(Slide.) The next slide presents the results of the cardiac catheterization studies by Dr. Henry Zimmerman of another patient with an atrial septal defect in whom evidence of reversal flow or, in other words, a right-to-left shunt could be demonstrated. This patient was a 13-year-old girl who had been cyanotic for one year. As you see, the pressure within the right auricle was 10 mm. of mercury and that within the left auricle was 5 mm. of mercury.

As experience increases, the lower level of arterial desaturation compatible with a successful outcome will be known.

DR. C. WALTON LILLEHEI, Minneapolis.—As has been indicated, a large number of methods are available for closure of atrial septal defects at the present time. However, none of these methods appears so safe or so satisfactory that one would care to recommend it for routine application to patients who are getting along quite satisfactorily, since obviously still better methods will become available in the very near future.

However, in those patients who have a large interatrial septal defect, and who are having trouble from congestive heart failure or from diminished cardiac reserve, it seems imperative to undertake surgical correction by one of the currently available methods. In choosing the best operation for such a patient there are several objectives that seem to us worthy of emphasis. The first of these is the necessity for closure under direct vision. I think that few surgeons in the audience will dispute the fact that palpation is frequently a poor substitute for vision, and thus, surgical procedures carried out under direct vision are far more likely to be satisfactory than those carried out blindly. This is particularly applicable to cases of interatrial defects.

In our own observations, a surprising number of these defects are multiple and lie scattered around the more inaccessible periphery of the septum and may be contiguous with the orifices of the venae cavae making blind closure hazardous of obstruction of these structures. Further, although occasional atrial defects are found in the center of the septum with a nice margin of tissue around them, unfortunately there is no way that I know of to predict such favorable types ahead of time. Moreover, other atrial defects are of the septum primum type in which the lower margin abuts directly upon the valve. In such a case if the closure procedure is done blindly, one of two complications is very likely to occur: either incomplete closure, or serious if not fatal interference with function of the atrioventricular valves.

And finally, as has been indicated today, many investigators have paid considerable attention to the development of various methods of closure of septal defects utilizing tissue grafts, autogenous or homologous, or prostheses of various kinds made of polythene or various other foreign substances.

Aside from the fact that the success of such methods is distinctly dubious, there remains the very important consideration that the use of such grafts or prostheses is quite unnecessary in closing either atrial or ventricular septal defects provided the heart is empty of blood at the time of closure.

These defects are best closed by direct suture of their contiguous tissue margins. In our observations, the edges of even the largest possible defects such as septum primum types literally fall together, if the stitches are pulled up or tied at a time when the heart is emptied of blood and thus its walls are flaccid. This is manifestly true, because the abnormal shunt has produced a compensatory enlargement of one or more of the adjacent chambers. In fact,

it is quite conceivable by consideration of Starling's law and other mechanical factors involved that the cardiac reserve would immediately benefit by having some of this "slack" taken up by direct suture at the time of closure of large defects.

With these criteria in mind, and with an attempt to retain simplicity, I should like to suggest, with some temerity, still another method for closure of atrial defects which fulfills some of these objectives although it is as yet untried clinically.

(Slide.) This method combines the use of an atrial well, as described by Gross for easy and repeated entrance into and out of the heart, together with intermittent vena caval occlusion to allow direct intracardiac vision in a relatively dry field.

In actual operation, the conical rubber well is sutured circumferentially to the right atrial wall. Tapes are placed around the superior and inferior venae cavae and then each is threaded through a short piece of rubber tubing. Quick and positive occlusion is obtained by pulling up on the tapes, and is maintained by crossclamping the rubber tubing with a hemostat.

(Slide.) Air embolism, in our experience, has been best controlled by threading a catheter into the left atrium through a pulmonary vein branch (this vein may be tied off without harm at the conclusion of the procedure). This catheter is connected to a pressure bottle containing sterile 5 per cent glucose for rapid filling of the left side of the heart. In addition a Satinsky clamp is placed through the transverse sinus, and the outflow tract of the heart is occluded proximal to the orifices of coronary arteries during the period the heart is empty of blood and contains air.

(Slide.) After preparations as indicated, the wall of the heart within the well is incised. Blood comes up within the atrial well to a height equal to the interatrial pressure. However, we customarily have used a well considerably more shallow than that described by Gross. Such a shallow well is techincally easier to work through, and any tendency to overflow during rest periods is readily controlled by partial vena caval occlusion.

Experiments in our laboratory (Surgical Forum 1952) have indicated that four minutes of complete vena caval occlusion is entirely safe in dogs. In actual practice we have thought it best to work two and one-half to three minutes; then if we are not finished with the suturing, the venae cavae are released after filling the left side of the heart with glucose from the pressure bottle and then releasing the outflow clamp. Two or three minutes are then allowed for the heart to recover and the entire procedure may be repeated.

By this method of intermittent vena caval occlusion with an atrial well, we have obtained as much as thirty minutes of working time within the heart in a dry field successfully in the dog. Obviously, most intracardiac procedures at present do not require nearly that much time.

DR. SHUMACKER (Closing).—I am sure we have all enjoyed the interesting discussions of Dr. Bailey, Dr. Kay, and Dr. Lillehei. They certainly demonstrate the variety of approaches which are presently being investigated and a certain degree of success which is being achieved. I think that it is still worth while to explore other methods in the search for the operation which may ultimately prove to be the safest and the best.

SURGICAL TREATMENT OF MITRAL INSUFFICIENCY

An Experimental Study

Max G. Carter, M.D.,* John M. Gould, M.D.** (by invitation), and
Bernard F. Mann, Jr., M.D.*** (by invitation)
New Haven, Conn.

INSUFFICIENCY of the mitral valve remains a major problem in the surgical treatment of valvular heart disease. A readily performed and physiologically sound corrective procedure is needed for those patients who are found to have mitral insufficiency as the predominant lesion. We have investigated the effect of a plastic prosthesis, placed in the left ventricular chamber, upon the pathologic physiology of mitral insufficiency.

MATERIAL AND METHODS

Experiments were conducted on twenty-six mongrel dogs which were subjected to thirty-eight operations. Early in the series there were six operative deaths associated with anesthesia and technical difficulties.

The experiments may be divided into two groups. In the first group, an effort was made to place a plastic ball encased in a pericardial tube between the mural leaflet and the endocardial surface of the lateral ventricular wall. There were ten of these experiments. In the first seven, the prosthesis occupied a poor position in relation to the mural leaflet or failed to penetrate completely the myocardium. With further experience it was possible to introduce the pericardial encased ball through the ventricular wall so that it consistently came to rest in the recess behind the center of the mural leaflet. The ventricular incision was made obliquely, in the direction of the muscle fibers, parallel and anterior to the descending portion of the circumflex left coronary branch, 1 cm. below the atrioventricular sulcus. With these landmarks as guides, subsequent mural leaflet balls were readily placed in proper position. The animals were sacrificed at postoperative periods ranging from two months to one year.

In the second group of eight experiments, a longer pericardial tube was fashioned allowing the ball to penetrate further into the ventricular chamber. This permitted the prosthesis to swing to and fro synchronously with the directional changes in blood flow, approximating a ball valve mechanism. This maneuver was performed in four animals as an acute experiment. It was performed in four other animals as a survival experiment under aseptic conditions, and these animals were sacrificed at intervals ranging from five days to two months.

From the Department of Thoracic Surgery, the Department of Pathology and the Surgical Research Laboratory, Hospital of St. Raphael.

This study was supported in part by a research grant from the Connecticut Heart Association.

Read at the Thirty-third Annual Meeting of The American Association for Thoracic Surgery, San Francisco, Calif., March 27, 28, and 30, 1953.

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We then attempted to produce mitral regurgitation and immediately to correct this defect by insertion of the pedicled ball valve. In some experiments regurgitation was produced by incision of either the mural or septal leaflet. In other cases the margin of the mural leaflet and its chordae tendineae were sutured to the ventricular wall. The degree of regurgitation was assessed both by the operator's finger within the left auricle and by pressure tracings obtained through a polyethylene catheter connected to a recording electromanometer. In one experiment regurgitation was created, the pedicled ball inserted, and the animal sacrificed at the end of five days. Initial exploratory cardiotomy in another animal revealed pre-existing mitral insufficiency. The pedicled ball valve was inserted and this animal was sacrificed at two weeks.

Electrocardiographic tracings were obtained on many of the animals before, during, and after surgery.

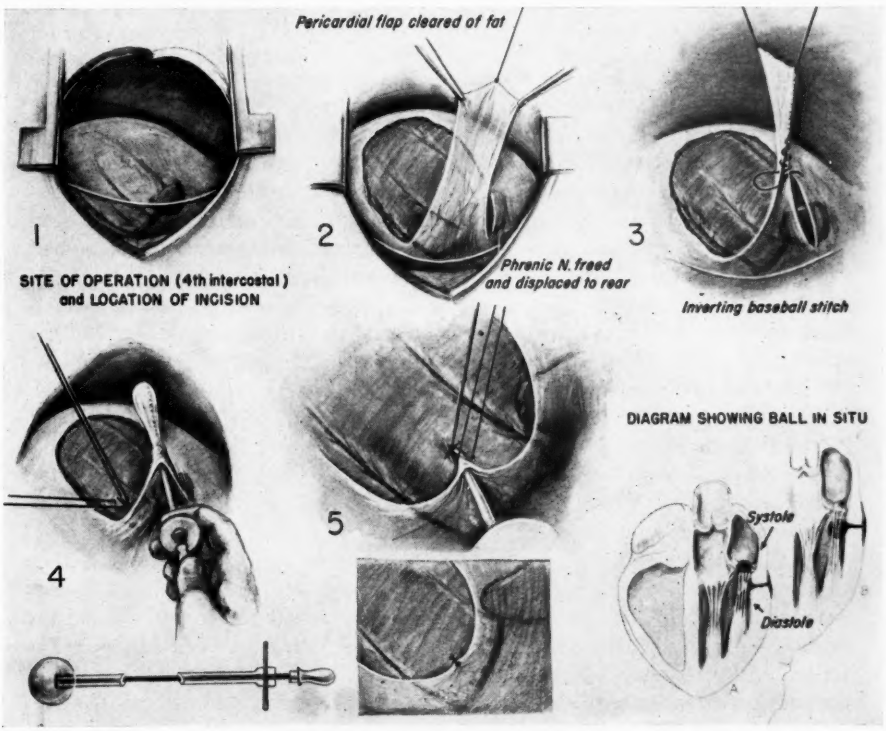

Fig. 1.—Artist's drawings of operative technique. Inset *A* (left) shows positions of pedicled ball during systole and diastole. Inset *B* (right) shows stationary ball as buttress of mural leaflet.

TECHNIQUE

The dogs were anesthetized with intravenous Nembutal, and intermittent positive pressure oxygen was administered by an endotracheal tube. The left hemithorax was entered through the fourth or fifth intercostal space and polyethylene manometric tubing was placed

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We then attempted to produce mitral regurgitation and immediately to correct this defect by insertion of the pedicled ball valve. In some experiments regurgitation was produced by incision of either the mural or septal leaflet. In other cases the margin of the mural leaflet and its chordae tendineae were sutured to the ventricular wall. The degree of regurgitation was assessed both by the operator's finger within the left auricle and by pressure tracings obtained through a polyethylene catheter connected to a recording electromanometer. In one experiment regurgitation was created, the pedicled ball inserted, and the animal sacrificed at the end of five days. Initial exploratory cardiotomy in another animal revealed pre-existing mitral insufficiency. The pedicled ball valve was inserted and this animal was sacrificed at two weeks.

Electrocardiographic tracings were obtained on many of the animals before, during, and after surgery.

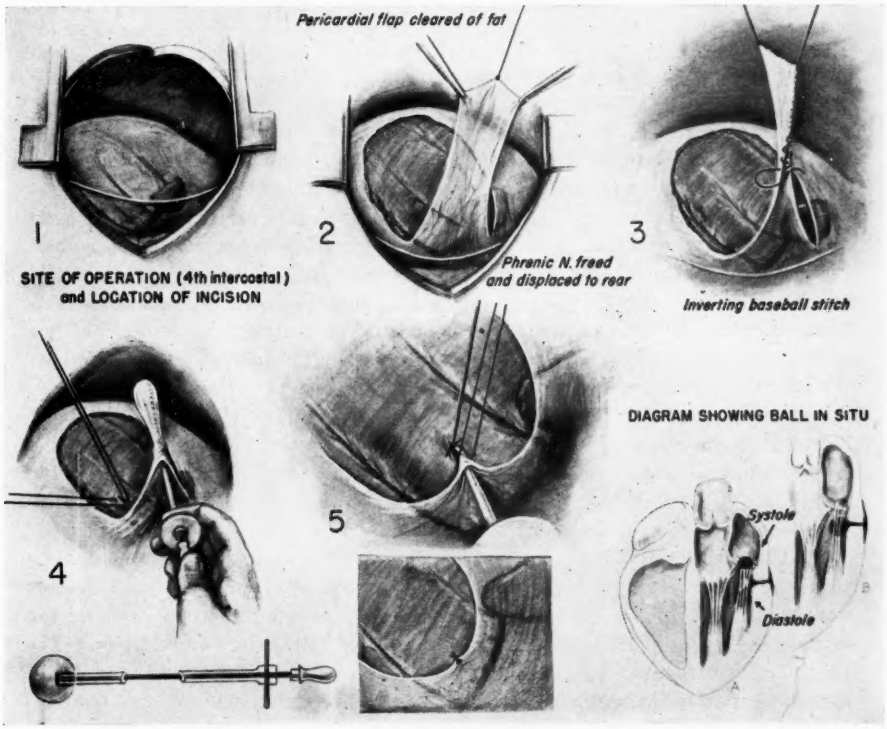

Fig. 1.—Artist's drawings of operative technique. Inset *A* (left) shows positions of pedicled ball during systole and diastole. Inset *B* (right) shows stationary ball as buttress of mural leaflet.

TECHNIQUE

The dogs were anesthetized with intravenous Nembutal, and intermittent positive pressure oxygen was administered by an endotracheal tube. The left hemithorax was entered through the fourth or fifth intercostal space and polyethylene manometric tubing was placed

in the left auricle through a small pulmonary vein. The phrenic nerve was freed from the pericardium and a rectangular pericardial flap was cut with its pedicle located at the base of the left auricular appendage (Fig. 1, *1* and *2*). This preserved the blood supply to the flap through its base since the pericardial vessels course obliquely in this area. This flap, measuring approximately 5 by 8 cm., was folded and closed to form a pedicled tube with the cardiac surface on the outside (Fig. 1, *3*). An inverting baseball stitch was used to minimize silk presenting on the surface of the tube. Two parallel sutures of 0 braided silk on atraumatic needles were then placed in the myocardium of the left ventricle at right angles to the atrioventricular sulcus, anterior to the circumflex branch of the left coronary in its descending course. The inferior ends of these sutures were tied and placed in a clamp held by the assistant. The nylon ball, impaled on the cannula type inserter, was placed in the pericardial tube.

A 1 cm. incision penetrating the ventricular chamber wall was made between the sutures in a direction parallel to the course of the myocardial fibers. The ball and pedicle were then inserted through the incision into the ventricular cavity and the remaining suture ends tied (Fig. 1, *4* and *5*). One or two additional myocardial sutures were usually required to achieve hemostasis. A palpating finger was then placed in the left auricular appendage using standard mitral valvulotomy technique and the position of the ball confirmed. The lung was expanded and the chest closed. Antibiotics were not used in these experiments. At sacrifice the intra-auricular pressure was recorded and digital palpation was performed.

RESULTS

In the early cases, where the short pedicled ball was placed as an immobile buttress behind the mural cusp, transauricular palpation confirmed the buttressing effect but there was no indication that the dynamics of the valve had been changed. Palpation in an animal with a longer pedicle revealed a type of ball valve action, the ball being in the mitral ring during ventricular systole, and in the ventricular chamber during diastole. When the pedicle was still longer, there was prolapse through the mitral orifice during ventricular systole. Neither mitral stenosis nor regurgitation was identifiable by the palpating finger following insertion of the ball in the normal heart.

Gross examination of the hearts after sacrifice confirmed the localization of the prostheses previously noted by the palpating finger. It was noted that the long pedicles allowed prolapse of the ball through the mitral orifice into the auricle (Fig. 2). No instance of intracardiac thrombosis, endocarditis, or rupture of a pericardial pedicle was observed.

After inspection of the removed hearts in the freshly opened state they were formalin-fixed in toto. Tissue blocks for microscopic study in all cases were cut from both mitral leaflets, the pericardial investment of the ball and the ventricular wall including the transmyocardial pedicle of pericardium. The following microscopic observations were made. There were no significant changes in valve leaflets. Myocardial healing had occurred with minimal reaction although foci of cartilaginous metaplasia were occasionally seen.

In all but one case, the pericardial pedicle had been converted into a dense collagenous scar. The pericardium investing the ball was thinned, collagenous, and practically avascular. The one exception showed a few endothelial-lined, blood-filled channels coursing longitudinally through the pedicle. The pericardial elements had retained their identity, but in its most dependent portion the pericardial investment of the ball was thinned to a degree where viability

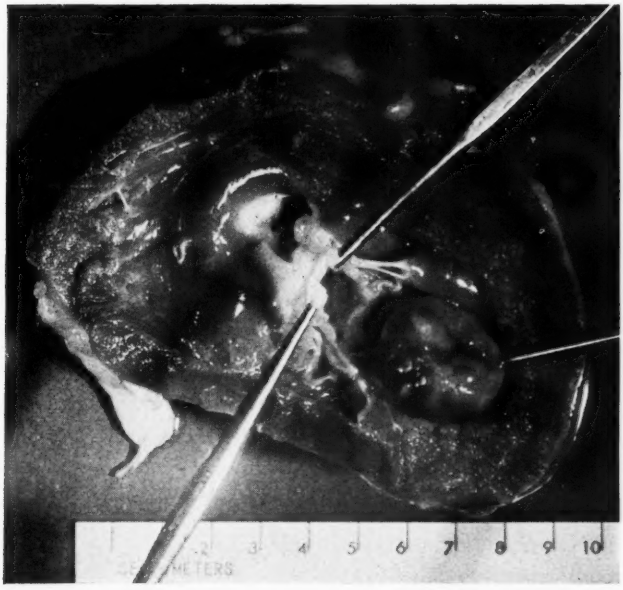

Fig. 2.—Photograph of heart opened through the tip of the left ventricle showing pedicled ball valve protruding below central portion of the mural leaflet. The aortic outflow tract at the left is separated by the septal leaflet from the mitral inflow tract at the right. An area of hemorrhage on the superior surface of the pericardial encased ball was the result of vigorous palpation at sacrifice (animal sacrificed at two months).

Fig. 3.—Photomicrograph (×6) showing pericardial covering at left with pedicle passing through ventricular wall at right. Note extreme thinning at far left and poor vascularity in the entire pericardial tube (animal sacrificed at two weeks).

might be seriously questioned (Fig. 3). These findings suggest that avascular necrosis, rupture of the sac and extrusion of the ball might have followed if the animals had been allowed to survive for a longer period.

Serial electrocardiograms revealed transient ventricular rhythms with S-T and T-wave changes during the operative and immediate postoperative periods. These findings were compatible with the pattern of pericarditis and with focal areas of myocardial damage. The electrocardiogram usually reverted to normal during the first postoperative week.

Neither mitral stenosis nor regurgitation was observed following insertion of the ball in the normal heart.

Intracardiac finger palpation revealed gross regurgitation after incision of either mitral leaflet. This was more marked when the septal leaflet was incised. Manometrically there were marked increases in the amplitudes as well as in the mean auricular pressures. Similar though less marked regurgitation patterns were obtained when sutures were placed through the ventricular wall to include the mural leaflet or its chordae tendineae.

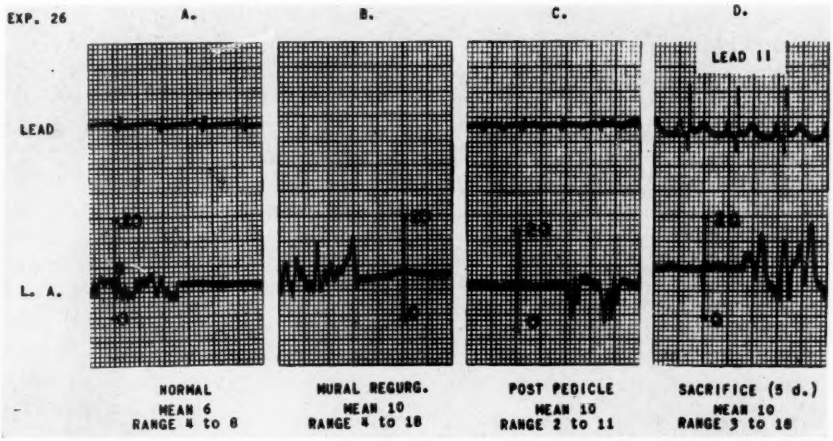

Fig. 4.—Simultaneous electrocardiographic and left auricular pressure tracings from Experiment 26. *A*, Normal preoperative findings with a mean intra-auricular pressure of 6 mm. Hg. *B*, Tracing after incision of mural leaflet creating palpable regurgitant jet with mean pressure measured at 10 mm. Hg. *C*, Tracing immediately after insertion of pedicled ball. Mean pressure remained at 10 mm. Hg. *D*, Tracings obtained at sacrifice operation five days later showed no change in pressures.

One animal demonstrated pre-existing mitral regurgitation at initial exploratory cardiotomy. This was identified by a systolic thrill over the left auricle and an easily palpable regurgitant jet. In spite of this jet, the mean auricular pressure was normal. This mean pressure was not changed by the pedicled ball valve although the shape of the pressure curve was modified. The jet, which was palpable before insertion of the prosthesis, was no longer present after insertion, nor was it present two weeks later at sacrifice, although the externally palpable thrill remained.

Mitral regurgitation, secondary to incision of the mural leaflet, exhibited a changed pressure pattern after insertion of the pedicled ball valve, although there was no change in either the mean pressure or the pressure range (Fig. 4). In this experiment, the palpable external thrill and the regurgitant jet were unaltered by the insertion of the pedicled ball valve. The jet and thrill were noted again at sacrifice, and palpation revealed the ball approaching the mitral orifice during ventricular systole but not prolapsing into the auricle. The ball was released from the overhanging mural leaflet by digital manipulation and allowed to prolapse into the auricle. This produced marked change in the pressure pattern and complete abolition of the palpable jet. The mean auricular pressure fell from 12 mm. of mercury to 8 mm. of mercury and the range from 13 mm. of mercury without prolapse, to 45 mm. of mercury with prolapse (Fig. 5). These changes were reversed by restoring the pedicled ball to its original position. The observations and tracings were confirmed by repeating the maneuvers several times.

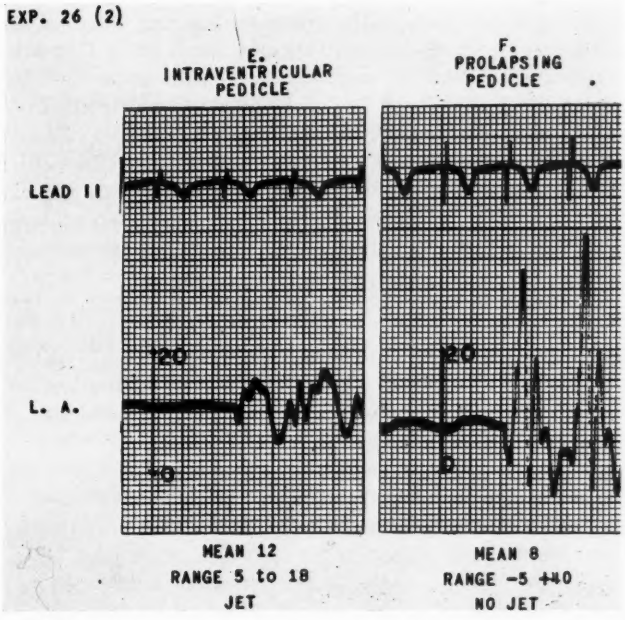

Fig. 5.—Continuation of simultaneous electrocardiographic and left auricular pressure tracings obtained at sacrifice operation in Experiment 26. *E*, A palpable jet was noted when the pedicled ball remained in the ventricular cavity and the mean pressure was 12 mm. Hg. *F*, Note reduction in mean auricular pressure to 8 mm. Hg and high pressure low volume waves of the prolapsing pedicled ball valve. Regurgitant jet was no longer felt.

DISCUSSION

Previous investigators have reported auricular wave patterns in experimental animals, normal human beings, and patients with mitral valve disease.[1-3] Quantitative evaluation of mitral regurgitation is difficult. Our own investiga-

tions suggest the inaccuracy of the palpating finger as a quantitative measurement of mitral regurgitation. This is implied by the observation of a palpable jet in one experiment which disappeared following the pedicled ball valve operation without an associated change in mean pressure or a significant change in pressure pattern. Other observers have noted that a large mitral opening during ventricular systole allows marked regurgitation with no palpable jet.[4, 5] However, it is frequently assumed that the degree of regurgitation is roughly proportional to the force of the jet. Our experiments cast doubt on this assumption.

These experiments further confirm the observations of Shumacker and his co-workers that autogenous tissue transplants such as vein, fascia, or pericardium are unsatisfactory for intraventricular use.[6, 7] We were disappointed that our pericardial pedicles showed early evidence of conversion to avascular collagen. In more than one instance there was sufficient attentuation of the pericardium surrounding the ball to suggest imminent rupture of the sac.

Nevertheless, our physiologic observations suggest that a moving prosthesis placed on the ventricular side of the mitral orifice may function as a tampon during ventricular systole to prevent regurgitation without creating stenosis during ventricular diastole. This suggests a rational basis for the "bottle baffle" operation of Harken.[8] We do not profess to have produced mitral regurgitation that is identical with that found in rheumatic heart disease. However, it does seem reasonable that mitral regurgitation which is physiologically similar to that found in rheumatic heart disease can be produced in the experimental animal. Valid observations can then be made which will permit development of a method for physiologic evaluation of human mitral regurgitation and the changes produced by operation upon the mitral valve.

SUMMARY

1. Plastic ball prostheses have been placed in the left ventricle adjacent to the mitral valve in a series of animal experiments. This has been done in normal animals as well as in animals with surgically produced, and in one case, naturally occurring mitral insufficiency.

2. No change was observed in the left auricular manometric pressure pattern of normal animals in which the plastic prostheses were used. Significant pressure and wave pattern changes were observed in animals with previously created mitral insufficiency following the insertion of pedicled balls.

3. The accuracy of intracardiac finger palpation in determining the volume of regurgitant flow is questioned.

4. The viability of an intraventricular pericardial pedicle is doubtful even when the pericardial flap is carefully cut to preserve its normal blood supply.

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DISCUSSION

DR. CHARLES P. BAILEY, Philadelphia.—We, too, have been interested in the problem of mitral regurgitation, either spontaneous or created in the course of performing mitral commissurotomy. I think if we could work under direct vision upon the incompetent mitral valve, every one of us would immediately suture together the incompetent portion which is usually located posteriorly. This can now be done by a closed technique.

(Slide.) The left index finger, ungloved, is introduced through the left atrial appendage to guide the passage of this special threaded suturing probe. The tip of the probe punctures the left ventricle at a point guarded by purse string and tourniquet. Guided by the intracardiac finger it punctures one valve leaflet at the point desired, follows the finger until it appears at the opening in the appendage so that one side of the suture can be grasped. That is withdrawn and the probe is pulled back. It is disengaged from this leaflet and is immediately redirected, punctures the opposite leaflet and again follows the finger. The opposite side of the suture must then be grasped, and then the probe can be withdrawn from the heart.

(Slide.) You see now that the double suture runs through both left heart chambers and each extremity pierces one of the valve leaflets in the usually posterior incompetent portion.

(Slide.) We now replace this silk suture with a strip of pericardium, a living suture, because we feel that the silk would eventually cut through the valve substance. By traction upon the ends of the silk suture emerging from the ventricle, we can then draw the loop of pericardium completely into the heart until the extremities appear out of the ventricular puncture wound. The finger can at once tell whether there is an appreciable change in the jet of regurgitant blood and frequently can recognize that it is completely abolished by this maneuver. If so, these pericardial ends are attached securely to the ventricular epicardium by sutures. If, however, there still remains appreciable regurgitation, the loop is converted into a noose by making an eye in one end and passing the other end through it. By pulling on the free end, this noose goes up to the valve and not only approximates but actually overlaps the two valve leaflets. Usually one such pericardial suture will greatly reduce the regurgitation, but if necessary a second can be placed. Obviously the free end has to be attached to the epicardium securely to maintain tension. If you reduce the opening too much, or have stenosis in association, a commissurotomy must be performed at the opposite anterior competent commissure.

We have had the opportunity of doing this on 70 or more patients since August, 1952. We have had a mortality, unfortunately, of approximately 25 per cent; this mortality rate was partly due to poor selection of very sick patients, and poor surgical technique during the developmental period. Recently, we have done very much better and have usually been able to completely or almost abolish the regurgitation without endangering the patient. We have had dramatic improvements in many of the patients who have survived. The pericardial graft I do not think does quite as badly as Dr. Shumacker has suggested in other conditions. After three or four months, if an autopsy is performed, the graft is seen to be pretty well welded and fibrosed to both leaflets so that they are glued together permanently. It does not seem as if the pericardium would dissolve.

DR. HARRISON BLACK, Boston.—There are several comments I would like to make on Dr. Carter's paper and the possible application of this method in human beings. This

report is based on work on Dr. Dwight E. Harken's service in Boston. The work began several years ago with animal experimentation at the Boston City Hospital and is being continued now with clinical material at Peter Bent Brigham Hospital.

Before considering the correction of any lesion we should know something about the pathology involved. We have therefore studied the lesion (mitral insufficiency) at the autopsy table in the museum, and at the operating table and have evolved a morphologic classification. On this first slide (slide) are represented the two primary types of *free* mitral insufficiency. Type I results from an absolute *loss of substance* due to scarring and shortening of the valve leaflets, predominantly the aortic, from direct rheumatic damage. There may be associated shortening of the chordae, but the predominant defect is insufficient leaflet length. Type II is the result of *dehiscence of the annulus* and consists of a relative loss of substance in the leaflets. This may occur due either to cardiac dilatation in failure or rheumatic carditis. It is probable that almost all cases of *free* mitral insufficiency fall into one or the other of these categories or the two may be combined, damage of leaflet preceding annulus dehiscence. It must be emphasized that all the patients upon whom we have operated for mitral insufficiency have been of these types of free insufficiency; there was not the slightest suggestion of stenosis. Furthermore, it is fundamental to an understanding of the condition to realize that in pure insufficiency as we have seen it, the leaflets are not calcified but are freely flexible. The defect is due solely to a failure in approximation.

(Slide.) This demonstrates three other types of insufficiency. Type III is the *directional* form resulting from a stenotic funnel facing out into the aortic outflow tract. Since the stenosis is predominant and the insufficiency relatively insignificant, correction entails merely the relief of stenosis. Type IV is due to *malocclusion* in heavily calcified, clumsy, stenotic valves in which irregularity of the leaflet edges prevents a competent watertight closure, and a ribbon-shaped regurgitant jet results. This may produce a noisy murmur, but it is not physiologically important and, again, correction of the predominating stenosis is the present treatment of choice. Finally, in Type V we find a *mixed type of process* in which there is the pathology of mitral stenosis in one area (leaflet fusion and/or calcification) while in another there is loss of substance without any trace of calcification. It is this type that is described by Dr. Bailey, if I understand his remarks about fracturing commissures before correcting insufficiency.

(Slide.) An understanding of the pathology of free mitral insufficiency makes it clear that adding substance to the defective leaflet has been the cornerstone of our corrective efforts. By such a method the mobile leaflets are given an opportunity to approximate something in systole and create a relatively sufficient valve. Dr. Carter has pursued a similar line of reasoning, but there are several reasons why we have discarded any thought of using the transventricular method of placement of the prosthesis. In this slide is shown in cross section the enormously dilated left ventricle we have encountered in cases of free mitral insufficiency. In the first eight patients we placed a Lucite ball beneath the ventricular leaflet via the auricular appendage and sutured it to the ventricular wall. We accepted only frankly terminal patients for this procedure, because we feel that in the developmental stages of any operation the surgeon is morally justified in accepting only this type of risk. These patients survived from one day to eight months, but it was obvious that not enough had been done to correct the insufficiency. At post-mortem examination all revealed that the prosthesis was well tolerated without damage to the myocardium, but because of the deep concavity beneath the leaflet formed by the dilated ventricular wall, the prosthesis was not close enough to the insufficient orifice to implement the loss of leaflet substance. Use of the approach through the ventricular wall would have the same defect, I believe, because it would be impossible in these immensely dilated hearts to place the sphere close enough to the valve orifice to be effective. The second objection to the transventricular approach is that in these dilated hearts the ventricular wall is thin and of poor quality due to congestive failure and chronic rheumatic carditis. In these we have often had difficulty controlling blood loss through the tiny needle sites in the myocardium and would despair of closing a hole large enough to allow the passage of a baffle.

There is one other point of fundamental physiologic importance that I should mention. If you will look at the sketch of the freely insufficient valve you will see that the flexibility of the valve leaflets allows them to balloon upward in a normal manner in systole. Although they do not approximate, the effective orifice is considerably narrower than in diastole when the leaflets wall aside and allow ventricular filling. This change in orifice size is an absolute necessity for the maintenance of forward flow of blood. If a fixed orifice exists or is produced by a technique which tenses the leaflets, the pressure gradients between ventricle and auricle would result in a lethal reversal of flow even if the heart rate were infinitely slowed and diastole infinitely prolonged.

The insert shows what we have done for the last twenty-one patients with free insufficiency. All were accepted for operation only because their outlook was considered hopeless on standard methods of medical management. A Lucite or nylon baffle, shaped like a bottle, has been placed beneath the minor leaflet and held firmly to the ventricular wall with one suture coming out above while the second comes out below the leaflet. The diagram illustrates the mobile aortic leaflet fallen aside during diastole allowing ventricular filling. In systole we have observed that the aortic leaflet approximates the baffle. That some correction has in fact occurred has been demonstrated several times by a rise of blood pressure by as much as 50 mm. Hg immediately after placing the baffle. The striking clinical improvement seen in several patients provides incontrovertible objective evidence that this effort is in the right direction.

We have recently had to reduce the size of the baffle since some have appeared to overcorrect the insufficiency as the annulus and ventricle have approached more normal size. There have been nine deaths. Three deaths were technical in nature. Three related to overcorrection by baffles that produced stenosis when the annulus came down in size in response to the correction of regurgitation. Two patients simply were too ill or too little benefited to survive, much less show improvement. One patient had embolization of the baffle eight months after operation. There are twelve patients surviving of the twenty-one who have had the present operation, and although these results are obviously not brilliant, they are sufficiently encouraging to justify further efforts.

DR. CARTER (Closing).—I have been fascinated, as I am sure you all have been, by the brilliant technical results presented by both Dr. Bailey and Dr. Black. I shall not argue the merits of each method. It does seem to me that the absence of an accepted standard procedure is at least some evidence that the final answer is not yet here. If I may be permitted the temerity of a suggestion, however, I personally would be much happier if instead of showing us human beings, they could show us well-documented long-term follow-ups on some animals, that would let us know exactly what is going on inside these hearts.

PULMONARY VALVULOPLASTY UNDER DIRECT VISION USING THE MECHANICAL HEART FOR A COMPLETE BY-PASS OF THE RIGHT HEART IN A PATIENT WITH CONGENITAL PULMONARY STENOSIS

F. D. Dodrill, M.D., Edward Hill, M.D. (by invitation), Robert A. Gerisch, M.D. (by invitation), and Aran Johnson, M.D. (by invitation)
Detroit, Mich.

BY CONGENITAL pulmonary stenosis is meant the stenosis of the pulmonary valve or infundibulum without overriding of the aorta. Tetralogy of Fallot is not, therefore, the subject matter of this manuscript. It is not an uncommon disease. Although the instance is not exactly known, the clinicians, who routinely carry out cardiac catheterization in congenital heart disease, are finding unsuspected cases.

The exact anatomic defect as well as the hemodynamics is a variable factor. The majority of patients with obstruction to the outflow of the right ventricle have defects in either the interatrial or interventricular septum, although there are some patients without other defects aside from the stenosis itself.

The stenosis may be purely valvular, in which case there is usually a protrusion of excess tissue out into the pulmonary artery, as is illustrated in Fig. 1. With cardiac catheterization, there is a very abrupt change in pressure at the valve area (Fig. 2). The stenosis also may be at the base of the valve cusp or in the infundibular region. Moreover, we have encountered patients with stenosis of both the valve and infundibulum. In such a patient, one finds with cardiac catheterization that the pressure is highest in the lower portion of the ventricle, with a much lower pressure in the infundibular region and finally a lower than normal pressure in the pulmonary artery (Fig. 3).

Some patients have been seen as infants, showing marked cyanosis, only to have this completely disappear at 1½ to 2 years of age. Concomitant with the disappearance of cyanosis, there has been a rapid increase in size of the right heart. Obviously such a change is due to a closure of a right to left shunt, most probably, patency of the foramen ovale.

With obstruction to the outflow of blood from the right ventricle and without a septal defect, there is usually a greatly elevated pressure in the right ventricle which is not uncommonly 175 to 180 mm. Hg or even higher. If there exists a septal defect, the pressure in the right ventricle is dissipated toward the left side of the heart, and there exists, therefore, a right to left

From the Research Division of Harper Hospital.
Aided by a grant from The Michigan Heart Association.
The Mechanical Heart used here was constructed by the Research Laboratories Division, General Motors Corporation.
Read at the Thirty-third Annual Meeting of the American Association for Thoracic Surgery, San Francisco, Calif., March 27, 28, and 30, 1953.

shunt. This produces arterial oxygen unsaturation which, if severe enough, is manifested by cyanosis, polycythemia, and mild clubbing of the digits. In some patients with pulmonary stenosis and a defect of the interventricular septum, the pressures may be essentially equal in both ventricle (Fig. 4). In such a situation, there exists a defect of the interventricular septum without significant shunting of blood through it, the pressures in the two ventricles having equalized. If there exists, therefore, no arterial oxygen unsaturation and if angiocardiography fails to show simultaneous opacification of the left heart and aorta, there exists no right to left shunt. Mild degrees of arterial oxygen unsaturation could be associated with a low pulmonary artery pressure.

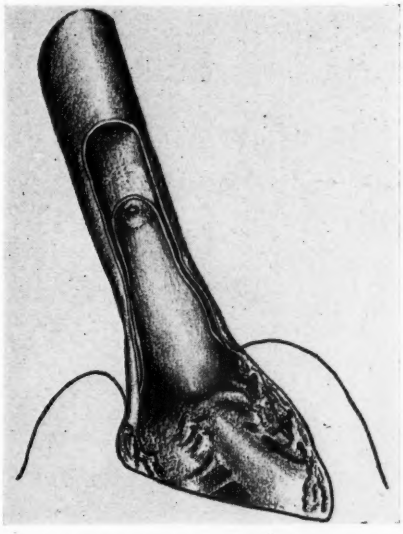


Fig. 1.—Diagrammatic sketch of congenital pulmonary bowel stenosis.

This does not necessarily mean that there is no defect present. We have observed one patient at autopsy a few years ago who had a slitlike opening in the interventricular septum which did not permit a right to left shunt, but would permit a left to right shunt provided the pressure in the right ventricle was lower than that in the left. This patient had been studied by cardiac catheterization. It showed a pressure of 140 mm. Hg in the right ventricle and an arterial oxygen saturation of 90 volumes per cent.

A blind procedure has been developed by Brock[1] for the relief of congenital pulmonary stenosis. This consists in inserting a valvulotome through the right ventricle and out through the stenotic pulmonary valve. Undoubtedly this is a good procedure and has vastly improved many patients throughout the world. There are some indications, however, that as time passes the pressure in the right ventricle may again become elevated.[2] Even though the pressure again becomes elevated, there seems to be clinical improvement of

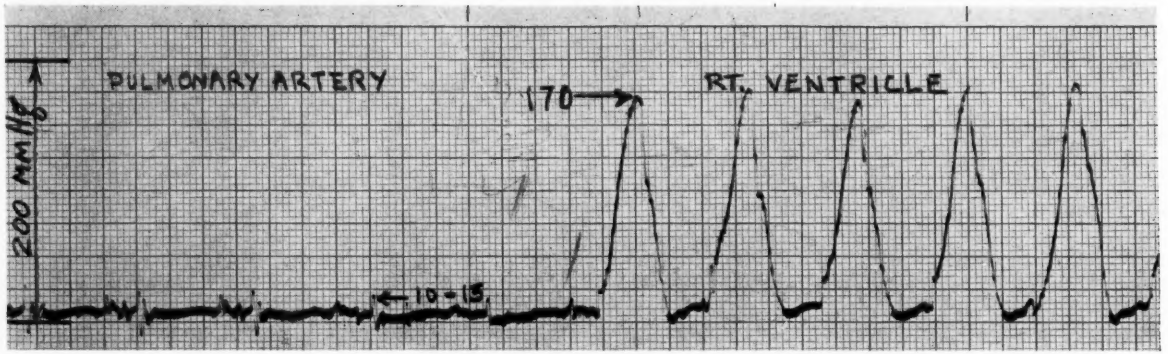


Fig. 2.—A high right ventricle and low pulmonary artery pressure seen in congenital pulmonary valve stenosis.

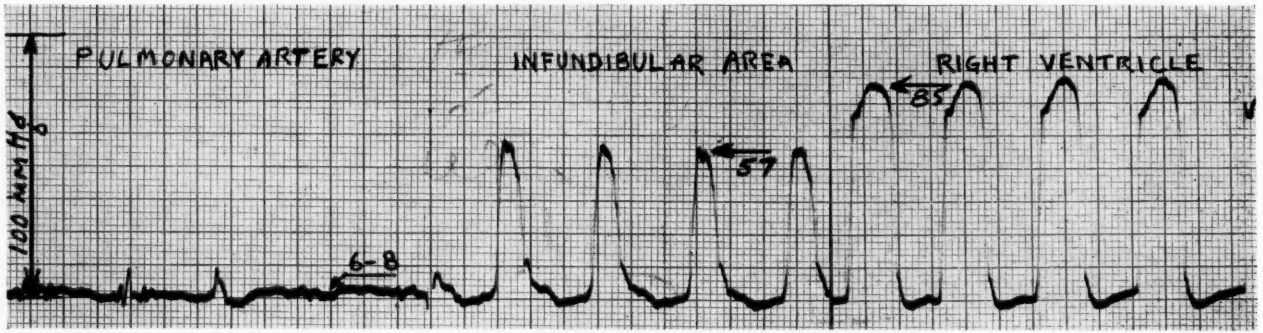
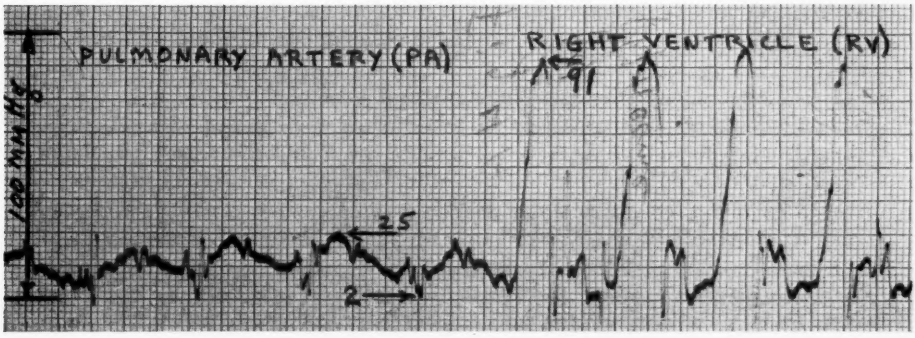


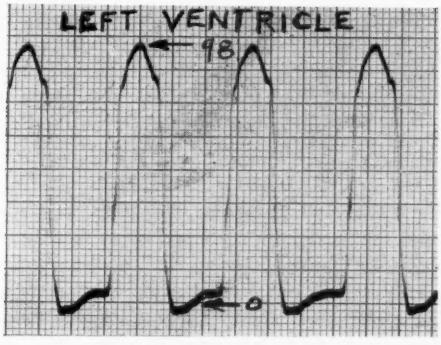
Fig. 3.—Illustrates both pulmonary valve and infundibular stenosis.

the patient. The elevation of the right ventricular pressure is not due to a left to right shunt through a possible interventricular septal defect, since this could be demonstrated by cardiac catheterization.

If the valvular architecture exists, as is illustrated in Fig. 1, and if such a valve is cut with the valvulotome, there is no assurance that the incision may not heal, producing the original obstruction. This is not definitely known.



A.



B.

Fig. 4.—Illustrates (*A*) a high right ventricle pressure and (*B*) a catheter was passed through an interventricular septal defect where arterialized blood was withdrawn. The pressure in the left ventricle is essentially the same as in the right.

More time must elapse and more cardiac catheterizations must be made on these patients one year or longer after the operation in order that it may be clarified. Although much good has been accomplished by blind procedures within the heart, it is probable that advancement from this point on must be made by exposure of the anatomic structure. With this thought in mind, the mechanical heart has been applied to a patient with congenital pulmonary stenosis. The right heart was completely by-passed. The main pulmonary artery was clamped off for twenty-five minutes while the left heart and lungs continued to perform their functions. The pulmonary valve was exposed and

enlarged under direct vision. The long flaps of valve tissue protruding into the pulmonary artery were excised.

We have had a great deal of experience with the mechanical heart in experimental work. There seems little doubt but that either the right or left heart can be by-passed while the other side and the lungs continue to perform their functions. With a by-pass of the right heart in dogs, the blood may be taken from the right atrium, passed through the mechanical heart and back into the pulmonary circuit through the artery to the right lower lobe of the lung, the cannula pointing centrally. By this method, blood is supplied to the middle lobe, the right upper lobe, and the entire left lung. The right ventricle and pulmonary valve thereby may be completely by-passed. Other investigators[3, 4] also have by-passed the right heart using similar methods in experimental animals. In the experimental work of this nature, we have found that with sufficient suction applied to the right atrial cannula the tricuspid valve apparently sealed itself and, although the right ventricle was opened, there was no bleeding or aspiration of air into the cannula. If there is a small amount of blood getting into the right ventricle, a light clamp may be placed across the pulmonary conus without interference with cardiac function. The pulmonary valve is, therefore, completely removed from the circulation. By this method, the coronary sinus blood, together with all the blood entering the atrium, is passed through the mechanical heart. If, on the other hand, the venae cavae are cannulated, there must be a separate plan for handling the coronary sinus blood.

The experimental work on dogs shows that there are right-sided hypotensive reflexes present which may be of extreme degree. These may be initiated by traction upon the pulmonary vessels.[5] In operations upon the lung on human beings, it is not usual to see significant hypotension while traction is being applied to the vascular structures. There could exist, however, short periods of hypotension in such operations on human beings which would not be detected by the usual sphygmomanometer method. This hypotension is thought to be due to the Bainbridge reflex. The hypotension due to traction upon the vascular structures may or may not be a part of the same mechanism. It is our impression that many physiologic studies need to be made on this problem.

CASE REPORT

The patient who underwent this procedure was a 16-year-old white boy. His weight was 76.3 kg. He had been short of breath on slight exertion for many years. The boy was unusually large for his age; he was well developed with large muscular structures. There was no visible cyanosis or clubbing of the digits, and the general physical examination was entirely normal except for the cardiovascular examination. The heart was regular in rhythm. There was a loud harsh systolic murmur over the pulmonary valve area. This could be heard less distinctly over the rest of the thoracic wall. The blood pressure was 110/70 mm. Hg.

The red blood count was 4,950,000 per cubic millimeter. There was 14.5 Gm. of hemoglobin per 100 c.c. The white blood count was 6,200 per cubic millimeter with a normal differential count. Urinalysis showed a specific gravity of 1.022 with no evidence of albumin, sugar, or cellular elements being present. The Kahn test for syphilis was negative. The nonprotein nitrogen was 30.0 mg. per cent; the prothrombin was 100 per cent

of normal; the clotting time was 13 minutes. X-ray examination of the thorax as well as fluoroscopy showed the right side of the heart, especially the right atrium, to be greatly enlarged. The main pulmonary arteries were hugely dilated. There were no visible pulsations of the pulmonary vessels seen at fluoroscopy.

The electrocardiogram showed marked right axis deviation and right ventricular hypertrophy. Cardiac catheterization revealed a pressure in the right ventricle of 190/0 mm. Hg. Preoperatively the pulmonary artery could not be entered. The pressure in the right atrium was 10 to 12 mm. Hg. Blood samples taken from the right ventricle, right atrium, and superior vena cava showed similar oxygen content, 15.87 to 16.83 volumes per cent. The arterial oxygen saturation was 90 volumes per cent.

Since this patient had a high ventricular pressure and since there was no cyanosis or significant arterial unsaturation, he obviously did not have a right to left shunt. The arterial oxygen saturation of 90 volumes per cent could be accounted for on the basis of low pulmonary artery pressure.

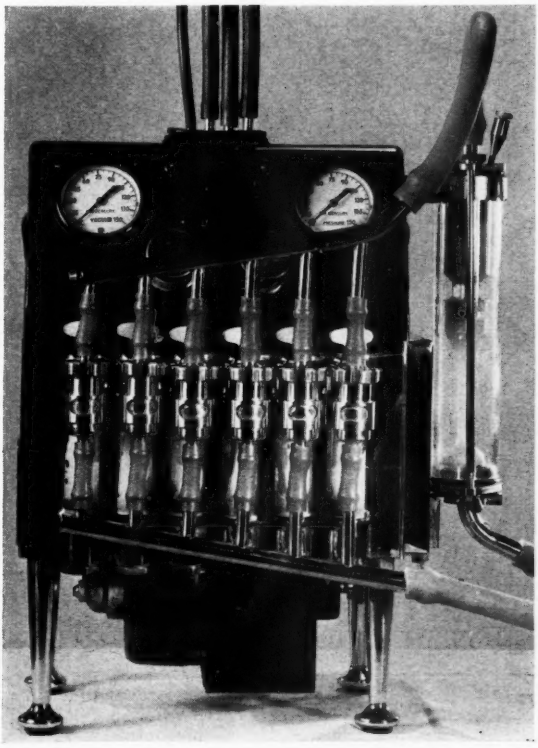


Fig. 5.—Photograph of the mechanical heart used.

Operation was carried out on Oct. 21, 1952. The mechanical heart and other apparatus had been assembled in the operating room before the patient arrived (Fig. 5). For a more detailed description of the mechanical heart used, the reader is referred to our earlier manuscript.[5] A large cannula was placed in the right ankle vein. The patient was then given 5 mg. of heparin per kilogram of body weight. A total of 38.1 mg. of heparin was used, and from this amount 8 mg. was placed into the donor blood which

filled the mechanical heart and the remainder was given intravenously to the patient. Ten minutes after the heparin was given, the clotting time was taken and this test was repeated for the next four hours, during which time there was no evidence of clot formation. The right ulnar artery was exposed, and a No. 8 Cournand cardiac catheter was inserted and passed up to about the innominate artery. This was connected to a direct recording device so that a continuous arterial pressure could be recorded at all times. A continuous electrocardiogram was also recorded (Fig. 6).

Ether anesthesia was administered by Dr. A. B. Stearns. A transverse incision was made on the anterior thoracic wall, and both thoracic cavities were entered through the third intercostal spaces. The sternum was transected. After inserting a rib spreader on either side, a very wide exposure of the thoracic contents was obtained. Inspection and palpation of the heart showed a huge right atrium and right ventricle. There was a very vigorous systolic thrill over the base of the pulmonary artery.

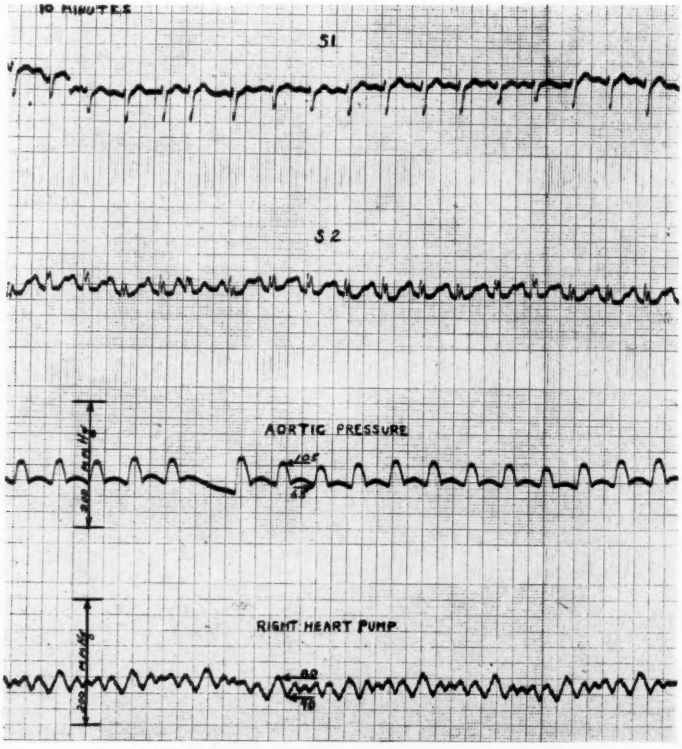


Fig. 6.—Record of electrocardiogram, arterial pressure, and the blood pressure as it leaves the mechanical heart during a complete by-pass of the right heart in a patient.

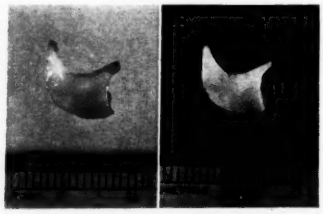
The interlobar fissure on the right side was separated, and the artery to the right lower lobe was isolated. An 8 mm. cannula was inserted into the artery and tied in place. The cannula was pointed centrally so that blood was supplied to the middle lobe, the right upper lobe, and the entire left lung. The pericardium was then opened on the right side, exposing a huge right atrium and atrial appendage. A 10 mm. cannula was inserted into the atrium through the appendage and sutured in place.

The mechanical heart already had been assembled and filled with donor blood. The tubing leading to the mechanical heart and away from it were then connected to the cannulae in the atrium and pulmonary artery, care being taken that no air bubbles got into the circuit. The mechanical heart then was slowly turned on until the entire right-sided circulation was diverted from the ventricle. The maximum flow through the mechanical heart was found to be 4½ L. per minute. The heart showed no arrhythmias and there was no great fall in systemic blood pressure. After the action of the heart and the electrocardiogram had been observed for two or three minutes, there being no change, the main pulmonary artery was isolated from the ascending aorta. It was greatly dilated. A clamp was placed across the pulmonary artery just proximal to its bifurcation. A light noncrushing clamp was placed across the pulmonary conus thereby completely walling off the pulmonary valve from the circulation. The stenotic valve which protruded out into the base of the pulmonary artery could be palpated. A transverse incision was made at the base of the pulmonary artery and the pulmonary valve was well visualized (Fig. 7).

Fig. 7.—Photograph taken at operation showing exposed stenotic pulmonary valve in a living patient. *A*, Retracted edge of incised pulmonary artery. *B*, Stenotic pulmonary valve.

A valvuloplasty was carried out under direct vision. A bicuspid valve was constructed and the long flaps of excess were excised (Fig. 8, *A* and *B*). The incision in the pulmonary artery was then sutured with No. 0000 arterial silk. The clamps were removed and the mechanical heart was slowly shut off, allowing the right ventricle to resume its former function. After the right ventricle had resumed its function, there was a very

faint systolic thrill felt in the dilated pulmonary artery. This could be accounted for by the dilatation of the pulmonary artery. The mechanical heart was disconnected from the cannulae which were then removed. The incision in the pulmonary artery to the right lower lobe was sutured transversely, re-establishing its continuity. The atrial appendage was ligated at its base and excised. The incision in the chest wall was then sutured with interrupted silk sutures with drainage tubes placed in both thoracic cavities. The heart action remained good throughout the procedure, there being no arrhythmias. There was considerable blood loss during the removal of the cannulae which produced moderate hypotension. The patient received blood replacement throughout the entire procedure.



A. *B.*

Fig. 8.—*A* and *B*, Flaps of excess pulmonary valve tissue excised.

Postoperatively the patient was in fair condition. There was some dyspnea, requiring oxygen therapy. The temperature remained elevated for approximately one week. The patient received intensive antibiotic therapy for three weeks. Blood-streaked sputum was raised for one week following the operation. X-ray examination of the thorax revealed bilateral lung disease which was interpreted as pulmonary edema. This completely cleared within two weeks. A low-pitched systolic murmur is still heard over the pulmonary valve area.

Extensive blood chemical examinations were made both before and after operation. No significant change could be detected other than those common to any major procedure of this magnitude.

The patient was discharged one month after the operation, having remained in the hospital longer than usual for purposes of observation. There has been marked clinical improvement, and the patient no longer complains of dyspnea. He has been working as a freight handler, although against advice.

The postoperative cardiac catheterization pressures are vastly different. Preoperatively the pressure in the right ventricle was 190 mm. Hg. Six months after the operation, the patient underwent cardiac catheterization, and the pressure in the right ventricle reached as high as 50 mm. Hg. The pulmonary artery pressure is now 22 mm. Hg (Figs. 9 and 10). Preoperatively it could not be obtained. The arterial oxygen saturation is now 97.2 volumes per cent, having risen from the preoperative level of 90 volumes per cent. It may be assumed that the lowering of the saturation was due to the low pulmonary artery pressure. The size of the heart has decreased as illustrated in Fig. 11, *A* and *B*. A photograph of the patient is shown in Fig. 12.

This is, to our knowledge, the first instance of a complete by-pass of the right heart in a patient. Obviously there must not be a defect of the cardiac septa with such a procedure. Dogliotti[6] reported a successful partial by-pass of the right heart of a patient undergoing surgery for a mediastinal tumor. He reported the blood flow through his artificial mechanism as 1 L. per minute. Since his patient was an adult, the amount of blood diverted from the right side of the heart would be approximately one-third to one-fourth of the total right-sided circulation. Since our patient did well with this procedure, it is proof that a unilateral by-pass of the heart in patients can be done. We rec-

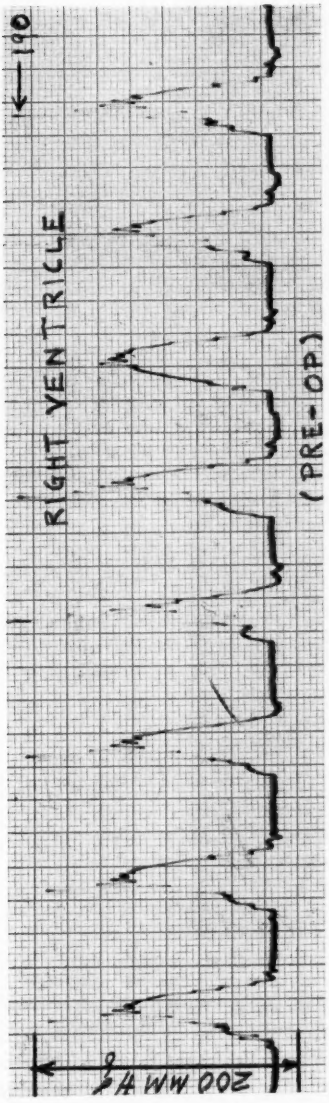


Fig. 9.—Preoperative right ventricle pressure in this patient.

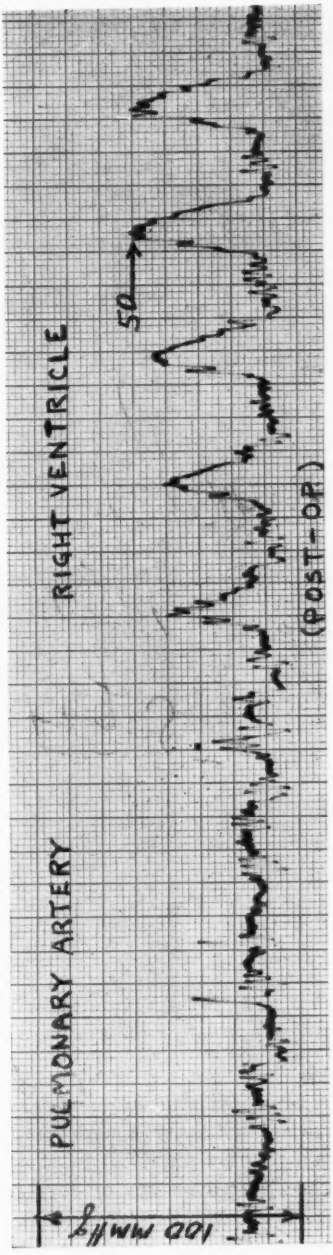
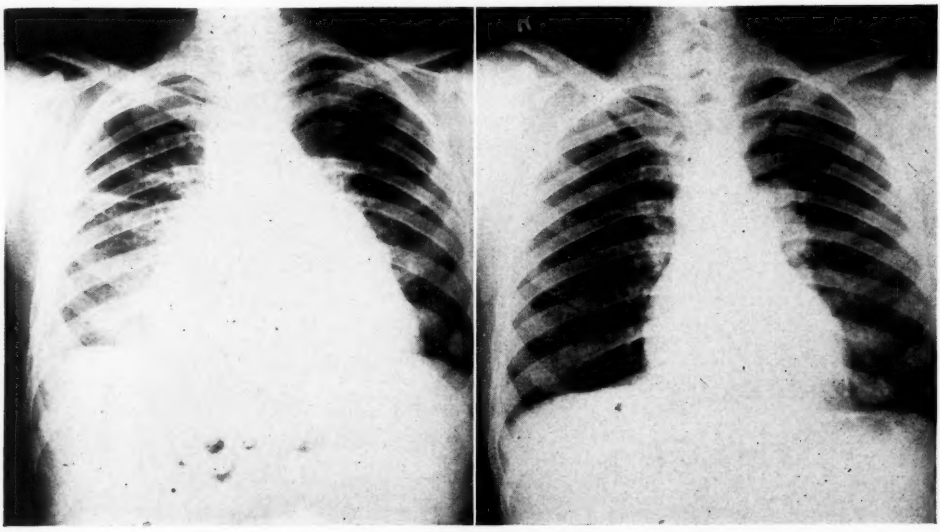


Fig. 10.—Right ventricle and pulmonary artery pressure six months after operation.



A. *B.*

Fig. 11.—*A*, Size of heart at operation; *B*, size of heart six months after operation.

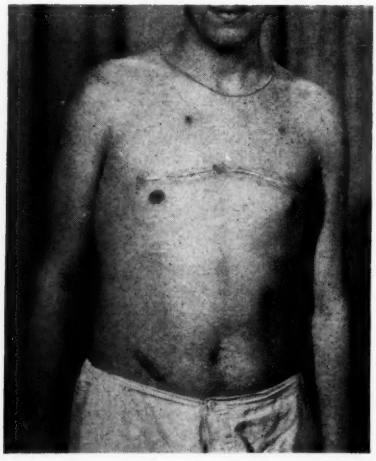


Fig. 12.—Photograph of the patient one month after operation.

ognize the fact that not many patients with congenital pulmonary stenosis could undergo this procedure, since many of them have septal defects as well.

There are not a great variety of diseases of the right heart in which the right-sided by-pass could be used. Pulmonary stenosis without other defects is one example. Diseases of the tricuspid valve may be another example. There are also rare examples of large emboli lodging in the right side of the heart producing serious warnings before a fatal issue.

Such a condition is briefly illustrated by the following experience. A young adult underwent surgery on the lower spine. During the postoperative

period, it was necessary that he be kept flat in a plaster cast. Approximately one week postoperatively, the patient became ambulatory and, as he was in the upright position, a very critical situation developed. He suddenly became extremely dyspneic and cyanotic. The attendants around him thought that he was dying. He collapsed and fell to the prone position, following which he revived and seemed essentially normal while in the prone position. The next day he was again made to sit up, and the same situation developed. He again collapsed and fell to the prone position, and was again revived. The following day he suddenly died. Post-mortem examination showed a huge embolus which had gotten into the right ventricle and would, in the upright position, totally obstruct the pulmonary valve. As the patient collapsed and fell flat, the embolus would drop down into the lower ventricle where the circulating blood could pass around it. If such a condition could be diagnosed in sufficient time, the embolus could be removed. These are mere examples of the application of a complete by-pass of the right heart, and probably many other conditions exist for the clinical application of this procedure.

SUMMARY

1. A successful complete by-pass of the right heart in a patient with congenital pulmonary stenosis is reported. The left heart and the lungs continued to perform their functions.

2. The main pulmonary artery was clamped off for twenty-five minutes while the pulmonary valve was exposed and enlarged under direct vision.

3. The blood flow through the mechanical heart was 4½ L. per minute.

4. The patient is vastly improved.

5. To our knowledge, this is the first successful total by-pass of the right heart in a patient.

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DISCUSSION

DR. CONRAD R. LAM, Detroit.—Apparently this rather large operation was utilized to correct valvular pulmonic stenosis because it appeared that the Brock operation is not a good one, although it was so referred to in the abstract. I am not aware that Mr. Brock has reported any unfortunate recurrences with his patients in which original satisfactory operations were performed. Our own experience has been with fourteen patients with valvular pulmonary stenosis, two of which were complicated. The twelve who had uncomplicated conditions are all well, with excellent postoperative catheter

studies. One of the two patients with complicated cases was an infant aged 3 months. The Brock valvulotomy was performed about ten minutes after the beginning of the operation. The child did not survive, and the autopsy showed that the aortic valve was involved in a similar process. The second patient who did not survive had a long infundibular stenosis in addition to the valvular stenosis, and there was difficulty in inserting the Brock valvulotome. Although the valve was opened moderately, for some reason death occurred several hours after the procedure.

The last operation of this type was on a 48-year-old woman. The total operating time, from the beginning until the incision was closed, was one hour and three minutes.

I should like to have Dr. Dodrill tell us if this is the only case he has had. One would suspect that a rather high initial mortality would ensue.

DR. HARRIS B. SHUMACKER, JR., Indianapolis.—Dr. Dodrill mentioned the need for more postoperative catheterization studies following pulmonary valvulotomy performed according to the method of Mr. Brock. Up to the present time there is very scant information of this sort in the literature. Although I have very little to add I thought it might be of some interest.

Dr. Paul Lurie and I have been interested in functional studies on patients with pulmonic valvular stenosis without ventricular septal defect. Thus far only seven patients have been subjected to catheterization before, and some time after, operation. All seven of these patients had mild or moderate symptoms. All have done very well clinically and have been relieved of the symptoms present before operation. Studies of right ventricular pressure after operation have been somewhat disappointing. In five patients there was a slight fall in right ventricular pressure and in two a marked fall, but in none was there a return to an absolutely normal level. The calculated valve area was the same after operation as before in two patients and was increased in the other five, but in none was it calculated to be more than three times as large as before operation. We realize, of course, the limitations of accuracy in the estimation of valve area.

Studies of arterial oxygen saturation, breathing room air and pure oxygen, have, on the other hand, been more encouraging. Of four patients who had a definite veno-arterial shunt before surgery, the evidence of shunt has completely disappeared in two and has been reduced to a borderline degree in the other two. Evidence of a borderline veno-arterial shunt in one patient has remained unchanged since operation. In another patient with an intermittent veno-arterial shunt before operation, no shunt is apparent afterward.

Possibly of greater significance than the other results of catheterization is the observation that one patient with a very high preoperative right ventricular pressure not reduced significantly by operation has been shown postoperatively to have a normal increase in cardiac output with exercise or, to put it more precisely, a normal increase in pulmonary artery flow. Quite in contrast, one patient with very mild pulmonary valvular stenosis and with a right ventricular pressure of only 40/0 demonstrates no rise whatsoever in pulmonary artery flow with exercise. She has not been treated surgically. It is possible that exercise effectively increases the mobility of the surgically made bicuspid valves and that the valve area is actually increased under stress over what it is at rest. It is planned to continue such studies.

We cannot help wonder whether the poor correlation between certain laboratory observations and the clinical course of the patients is not due to the fact that, up to the present, we have not focused our attention upon those particular laboratory studies which would reveal an actual improvement comparable to that which has been observed in the clinical course of all our patients, regardless of whether their symptoms before operation were mild, moderate, or marked.

DR. HENRY SWAN, II, Denver.—We too have been much concerned about the tendency of the right ventricular pressures to stay well above normal limits following the Brock procedure, in spite of the fact that there is marked clinical improvement in the patient. For that reason we have considered the possibility of actually excising the valve, realizing of course that this would cause regurgitation. Dr. Hufnagel has shown that one can excise the pulmonary valve in the normal dog. In addition, on theoretical grounds

we felt it might be feasible because the regurgitant pressure would presumably amount to approximately only 8 mm. Hg, that is, the diastolic pressure of the pulmonary artery. For that reason we have recently given this a clinical trial in three patients by the use of general hypothermia.

(Slide.) It is necessary to have a bathtub full of ice water to put the patient in, with a constant temperature recording machine. Note the anesthetist's respiration device; hyperventilation throughout the procedure is very important.

(Slide.) The operative procedure is essentially simple. One puts tapes around the venae cavae and ties the azygos vein. Pull up on the tapes and after about thirty to forty-five seconds the heart will be quite empty. It is now possible to make an incision in the pulmonary artery directly over the valve, and in the dry field there it is possible to grasp the valve and excise it. A nonerushing clamp is then applied to the incision in the pulmonary artery after flooding it with saline solution. Blood flow may now be resumed while the artery is sutured. We actually occluded the circulation in these three patients, one for seven and one-half minutes, one for three and one-half minutes, and one for three minutes and five seconds.

(Slide.) This shows the valve resected in one patient, and includes the original small lumen. Here is the valve with the lumen.

In these three children, all had essentially a noneventful postoperative course. The period of observation has been short. They had immediate dramatic improvement in exercise tolerance, but I would not like anyone to believe that at this time we recommend this procedure for general adoption. Certainly we must wait for a time to evaluate the effect of total valvular excision and the importance of pulmonary regurgitation in the human patient.

DR. GEORGE H. HUMPHREYS, New York.—I am surprised to hear of these very radical operations for an anomaly which can be corrected as directly and simply as it can be by the Brock procedure. In an experience of about thirty cases, using the conventional Brock knife, we have had only one death, and that was due to a technical error. I think that the degree of right ventricular hypertension which persists after the procedure is not very well known, and probably some of those patients in whom it has been documented have had a high right ventricular pressure ever since the procedure; in other words, the procedure has not been an adequate one to begin with.

We have been able to catheterize a number of these patients on the operating table and obtain right ventricular pressures immediately after the Brock procedure. In some cases when we felt that technically we had done a good job, we found that the pressure was still high, and we were able to go back and stretch the valve again and obtain a normal pressure. I think that perhaps some of the late so-called poor results are due to an inadequate operation in the first place. I think another cause is often overlooked, that it is a very small ventricular septal defect. Several of these have been encountered that were not detected by catheterization before operation. In these patients the right ventricular pressure balances the left, and there is little or no shunt in a high small ventricular septal defect, that is, no arterial blood will be detected in the right outflow tract on catheterization. However, after operation, arterialization can be detected in the outflow tract, and that means that a small ventricular septal defect is present and that the high right ventricular pressure is due to this and not due to recurrent stenosis.

DR. DODRILL (Closing).—I realize that a right-sided by-pass will not be applicable to many patients with congenital pulmonary stenosis because the vast majority do have septal defects of one sort or another. The point that this illustrates is that a right-sided by-pass seems to be a practical procedure that may be applied in the future to other diseases, aside from pulmonary stenosis. I think such drastic procedures as those that are in a development stage will of necessity carry a higher mortality rate than those procedures which have already been worked out. When we think back a few years to recollect the mortality rate in simple lung resections, then a mortality rate of less than 50 per cent, which we have had in our over-all results, I think is not too discouraging, particularly in view of the fact that most of the patients have been far advanced, and may possibly have carried a similar mortality with the Brock procedure.

THE PRODUCTION AND REPAIR OF INTERATRIAL SEPTAL DEFECTS UNDER DIRECT VISION WITH THE ASSISTANCE OF AN EXTRACORPOREAL PUMP-OXYGENATOR CIRCUIT

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PHILADELPHIA, PA.

A NUMBER of methods have been proposed and employed for the closure of interatrial septal defects without diverting the circulation from the heart. In none of these methods can the surgeon see the margins of the septal defect.

Cohn[5] demonstrated that experimentally produced defects in the interatrial septum could be closed by suturing the wall of the right atrium to the margins of the defect. The stalk resulting from the invagination of the right atrial wall was then divided and the defect in the atrial wall sutured. Bailey[2] has recently modified this procedure in patients by not dividing the invaginated stalk. He also inserted a finger into the right atrium during the procedure so that the margins of the defect could be palpated during the placement of sutures. With large defects, unless the atrium is greatly dilated, there is some danger of obstructing the return from the venae cavae by this method. Shumacker[18] has avoided much of the reduction in size of the atrial chamber by the use of a pouch of pericardium. A small sac of pericardium is constructed and sutured to the margins of an incised wound in the atrium. The sac is then invaginated into the atrial chamber and sutured to the margins of the atrial defect.

Swan and his associates[19] have closed experimentally produced interatrial septal defects in animals by invaginating both auricular appendages and opposing the tips through the septal defect by mattress suture through two plastic buttons. A modification of this method has recently been applied to the closure of interatrial septal defects in human patients.[20] Postoperative studies in two patients showed incomplete closure of the defect.

Murray,[16] in four human beings, attempted to close defects in either the interatrial or interventricular septum by passing mattress sutures of fascia lata or silk across the defect in the plane of the septum. When these sutures were tied, the resulting compression of the septum apparently reduced the size of the defects. No subsequent reports have appeared and it seems unlikely that complete closure of septal defects can be accomplished by this method.

Large experimentally produced defects in the interatrial septum have been successfully occluded by means of a plastic prosthesis. Hufnagel and Gillespie[10]

From the Department of Surgical Research of the Jefferson Medical College.
This investigation was supported by a research grant from the National Heart Institute of the National Institutes of Health, Public Health Service.
Read at the Thirty-third Annual Meeting of The American Association for Thoracic Surgery, San Francisco, Calif., March 27, 28, and 30, 1953.

introduced plastic discs on either side of atrial septal defects and then mechanically secured them to each other so that a rim of septal tissue was grasped by the discs. These plastic prostheses were rapidly covered with endothelium and were apparently well tolerated by experimental animals. In order to use this method, the rim of septal tissue must be complete, otherwise the button will not remain in position.[8]

Gross[8, 9] has closed interatrial septal defects in seven patients, operating through an open rubber well sutured to the margins of a wound in the right atrium. Blood rises a few centimeters in the well to a height equal to the atrial pressure. By digital palpation through the well, Hufnagel buttons were placed across the defect in three patients, a sheet of nylon or polyethylene was sutured to the margins of the defect in three other patients, and in one patient two defects were closed by simple suture. Because of incomplete septal rims the Hufnagel buttons became dislodged and the three patients subsequently died. One death occurred in the three patients whose defects were closed by nylon or polyethylene sheets. The three surviving patients are all improved but catheterization studies reveal a persistent but greatly diminished left-to-right shunt at the atrial level.

In the methods just referred to the surgeon has the serious handicap of being unable to see the extent of the defect or the operative field in which he is working. A clear field of vision can be obtained only by preventing the blood in the venae cavae from entering the right atrium. Without an extracorporeal blood circuit to maintain cardiorespiratory functions, the venae cavae can be safely occluded for only three or possibly five minutes. Lowering the body temperature to reduce metabolism has recently been proposed and employed to increase the duration of safe caval occlusion.

Martin and Essex[13] and Swan[19] have experimentally created interatrial septal defects under direct vision during exclusion of the heart from the circulation by caval occlusion at normal body temperatures. By reducing the body temperature, Bigelow[3] has shown that the period of caval occlusion may be markedly prolonged. In a recent article,[4] Bigelow reports that with hypothermia in monkeys, the heart may be excluded from the circulation for as long as twenty-four minutes with survival. This method has been used successfully in human beings.[1, 12] Without extracorporeal maintenance of the cardiorespiratory functions, caval occlusion, even with hypothermia, provides only a brief safe interval of time for open cardiotomy. Exceeding this brief time interval will result in cerebral damage or death.

In our opinion a safer method of obtaining a bloodless heart consists in the maintenance of the cardiorespiratory functions of the subject by a pump-oxygenator apparatus while the venae cavae are occluded. This gives adequate time for precise surgical procedures within the open heart. Both venae cavae have been occluded in animals for as long as one hour and forty minutes with prolonged survival of the animal in a healthy condition.[14] Dennis and his coworkers[6] attempted the repair of an interatrial septal defect in a human being under direct vision, using an extracorporeal circuit embodying an oxygenator. The defect was closed but the patient died.

Any method which exposes the left side of the heart to atmospheric air (either a left cardiotomy, or a right cardiotomy in the presence of a septal defect) carries with it the danger of air embolism in the systemic arteries. Martin and Essex[13] and Swan[19] observed air embolism in the coronary arteries during the production of interatrial septal defects. The hazard of air embolism in the systemic arteries during the production or repair of interatrial septal defects in the opened heart still exists in hypothermic states. Lewis and Taufic[12] filled the left atrium with physiologic saline solution while the aorta and coronary arteries were clamped during closure of an interatrial septal defect in a human being. The clamp was released after the wound in the atrium had been sutured.

This method of avoiding air embolism by attempting to keep the left cardiac chambers full of fluid is not uniformly successful. Senning[17] has demonstrated in dogs that air embolism of the systemic arteries can be avoided by inducing ventricular fibrillation during repair of atrial septal defects under direct vision employing an extracorporeal blood circuit. The pumping action of the heart is arrested during ventricular fibrillation and air cannot be pumped into the aorta. After the atrial wound had been sutured, normal rhythm could be re-established by a defibrillating electric shock.

The following report demonstrates that in animals interatrial septal defects can be successfully created and repaired under direct vision with an open cardiotomy while the cardiorespiratory functions are maintained by a mechanical heart and lung apparatus during the period of caval occlusion. In addition, the danger of air embolism can be avoided by what appears to be a safe and practical method.

APPARATUS

The pump-oxygenator used in these experiments is basically the same as that previously reported,[15] but the method of withdrawing blood from the venae cavae has been modified. Previously the roller type pumps were connected directly to the cannulae in the venae cavae. At the beginning of each pumping cycle when the roller began to compress the rubber tubing, there was a momentary positive pressure in the cavae. This was immediately followed by negative pressure as the roller moved onward compressing the tubing through an arc of 180 degrees. During the negative pressure phase of the pump cycle, the venae cavae frequently collapsed at the orifice of the cannula. Obviously the peak negative pressure of a fluctuating pressure cycle will be higher than a uniform negative pressure producing the same flow rate. Thus a lower steady negative pressure should produce the same rate of blood flow with less fluttering and intermittent occlusion of the cavae. By interposing between the cannula and the pump a vacuum chamber in which the blood collects, the pulsations produced by the pump have been eliminated.[7, 11]

The modified extracorporeal circuit is illustrated diagrammatically in Fig. 1. The blood from both venae cavae is carried through a Tygon tube to the collecting chamber (*A*). A slight negative pressure is maintained within this chamber, which is placed at approximately the same level as the venae cavae. As blood from the cavae enters the collecting chamber, the level of the blood rises, thus

automatically energizing the pump (*E*). The pump (*E*) automatically maintains a constant level of blood in the negative pressure collecting chamber (*A*) by means of the variable capacitor previously described.[15] With the smooth constant suction obtained by the interposition of chamber (*A*) between the roller pump and the vena cava, individual control of the flow rate from each cava is no longer necessary.[14] The amount of negative pressure needed to empty the cavae can be easily and quickly regulated. The negative pressure required is rarely greater than 10 mm. mercury.

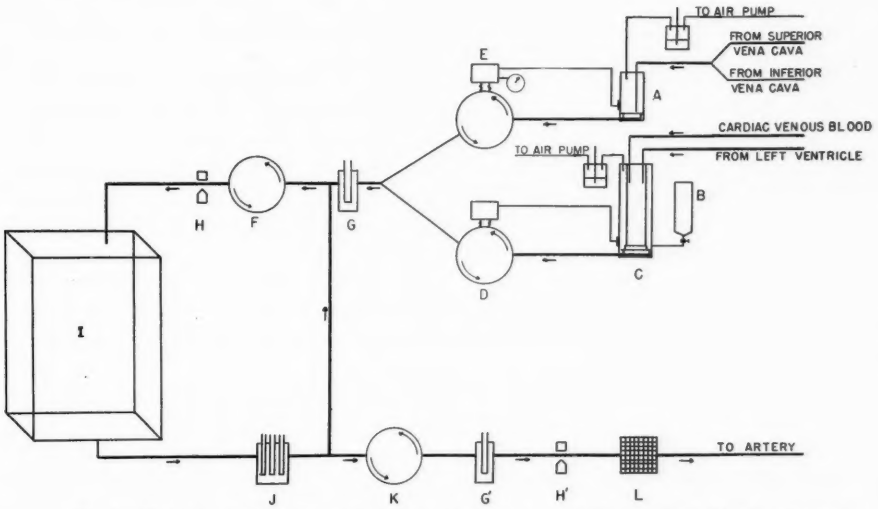


Fig. 1.—Extracorporeal pump-oxygenator circut. *A*, Systemic venous blood collecting chamber. *B*, Blood burette, *C*, Collecting chamber for blood from open right atrium and blood from vent in left ventricle. *D*, Automatically controlled pumps for emptying cardiac venous blood collecting chamber. *E*, Pump for emptying venous systemic blood collecting chamber. *F*, Oxygenator pump. *G*, Thermocouple for venous blood temperature. *G'*, Thermocouple for arterial blood temperature. *H*, High-pressure sensing device. *H'*, High-pressure sensing device. *I*, Screen oxygenator. *J*, pH electrode holder. *K*, Automatically controlled arterial pump. *L*, Filter.

The cardiac venous blood entering the right atrium must be continuously removed from the operative field in order to provide exposure of the interatrial septum. The blood and air aspirated from the right atrium are conducted through a Tygon tube to the inner cylinder of collecting chamber (*C*) and spread in a thin film.[14] The bubbles of air disperse from the blood film as it descends by gravity to the pool at the bottom of the chamber.

As stated, when an open right atrial cardiotomy is performed in the presence of an interatrial septal defect, the left atrium is exposed to atmospheric air. During diastole, air and blood pass through the mitral valve into the left ventricle. During systole the mitral valve closes, and both air and blood will be forced into the aorta with resultant air embolism of the coronary and other systemic arteries. The simplest and safest method of avoiding such air embolism is to provide a low-pressure vent for the left ventricle. With systole, the contents of the left ventricle will then be expelled through the vent instead of

being forced into the aorta. A Tygon tube 5 mm. in diameter is inserted into the left ventricular chamber by means of a stab wound in the apex. This tube conducts blood and air from the left ventricle to the collecting chamber (*C*). The negative pressure in chamber (*C*) is maintained at approximately 30 mm. Hg.

In the following experiments the blood collecting in the bottom of chamber (*C*) was returned to the venous side of the extracorporeal blood circuit by means of a manually controlled pump. As illustrated in Fig. 1, a variable capacitor, similar to that operating pump (*E*), is being constructed which will automatically energize the pump (*D*) and maintain a constant blood level in the chamber (*C*). This has been shown to operate satisfactorily.[14]

The oxygenator pump (*F*) draws blood from the bottom of the oxygenator case as well as from the two venous pumps (*D*) and (*E*). The flow through this pump is fixed at a rate which is greater than the sum of the maximum flow rates through the pumps (*D*) and (*E*). The oxygenator pump maintains a constant flow rate over the screens and so insures constancy of the volume of blood held on the screens. This avoids any change in the volume of blood in the subject's blood vessels during the course of the perfusion.

The high-pressure sensing device (*H*) becomes operative in the event of occlusions of the slits in the distributor chamber of the oxygenator and stops all pumps in the extracorporeal circuit. This event has never occurred in eighteen months' continuous use of the apparatus.

The oxygenator (*I*) consists of a series of vertically suspended stainless steel screens.[15] Blood enters the distributor chamber at the top of the oxygenator and then descends by gravity over the screens and accumulates in a small pool at the bottom of the oxygenator case. An electronic device similar to that controlling the pumps (*D*) and (*E*) operates the arterial pump (*K*) so as to maintain a constant level of blood in the bottom of the oxygenator case at all rates of blood flow.

Before entering the subject, the oxygenated blood passes through the electrode assembly (*J*) where the pH is continuously measured and recorded by a self-balancing potentiometer. The pH is automatically maintained at the level desired by the intermittent addition of carbon dioxide to the oxygen flowing through the oxygenator. Thermocouples placed within the chamber (*G*) and (*G'*) measure the temperature of the venous and arterial blood. The temperatures are recorded on a self-balancing potentiometer on the front panel of the main cabinet. The high-pressure sensing device (*H'*) automatically stops the venous and arterial pumps should high pressure develop as a result of an obstruction in the tube returning blood to the subject. The pumps remain inoperative until the obstruction has been relieved.

PREPARATION OF THE APPARATUS

The apparatus was prepared as has been previously reported.[15] All components of the blood circuit were completely disassembled and mechanically cleaned with detergent solution. Following reassembly of the apparatus, the blood circuit including the oxygenator case was filled with a 1:1,000 solution of

aqueous benzalkonium chloride. After one hour the benzalkonium chloride solution was removed and the blood circuit filled with sterile physiologic saline solution. The saline solution was then drained and the system washed with six separate fifteen-minute rinses of 1,500 ml. of sterile physiologic saline solution.

The tubing of the extracorporeal circuit was then filled with heparinized blood obtained from donor animals. These animals received an intravenous injection of 3 mg. per kilogram of body weight of heparin before exsanguination. Usually 2,000 ml. of heparinized blood was obtained from donor animals. The extracorporeal circuit volume was 1,200 ml. when the small oxygenator was used. An additional 600 ml. of heparinized blood was required when the large oxygenator[14] was used. The blood remaining after the extracorporeal circuit was filled was used to replace blood lost during the operation and also blood withdrawn for gas analysis. An additional 1,000 ml. of citrated blood was obtained from a donor animal. This was used to replace blood lost by serosanguineous effusion into the pleural cavities postoperatively.

The donor blood was circulated through the apparatus from a small flask in a constant temperature water bath. The blood film was established on the screens of the oxygenator a short time before the subject was connected to the extracorporeal circuit. The oxygenator pump (*F*) was set at a flow rate of 1,800 ml. per minute when the small oxygenator was used and at a rate of 3,500 ml. per minute when the large oxygenator[14] was in the circuit.

OPERATIVE TECHNIQUE

The dog was given an intramuscular injection of 3 mg. per kilogram of body weight of morphine sulfate approximately forty-five minutes before operation. Anesthesia was induced and maintained by intravenous 2.5 per cent Pentothal Sodium solution. An endotracheal tube with an inflatable cuff was placed within the trachea and connected to an electrically controlled positive and negative pressure respirator capable of producing apnea by overventilation.

Both femoral arteries were exposed. With the animal lying on its left side, the right pleural cavity was entered through the bed of the fifth rib. The superior and inferior venae cavae and the vena azygos major were exposed and braided silk ligatures placed beneath them. Heparin was then injected intravenously in the amount of 3 mg. per kilogram of body weight. The arterial and venous pumps were stopped, but the oxygenator pump maintained the blood flow over the screens of the oxygenator. Clamps were applied to the tubes leading to the extracorporeal circuit. The arterial and venous cannulae were then connected to these tubes. Generally one, but occasionally both, femoral arteries were cannulated centrally for the return of arterial blood from the apparatus. Either the remaining femoral artery or the left brachial artery was cannulated for direct measurement of blood pressure.

One venous cannula was inserted in a cephalic direction in the superior vena cava through an opening in the azygos vein. A pedicled pericardial graft was then cut (Fig. 2, *A*). The attached base of the graft was cephalad. The second venous cannula was then inserted in the inferior vena cava through an opening in the tip of the right auricular appendage.

The clamps were removed from the tubes leading to the extracorporeal circuit, thus diverting a part of the systemic venous return to the heart through the apparatus. The arterial pump automatically returned, at the same rate, arterial blood to the femoral artery from the extracorporeal circuit. The ligatures previously placed around the venae cavae were tied over the cannulae completely diverting the systemic venous blood flow to the extracorporeal circuit (Fig. 2, *B*). Ventilation of the lungs by the respirator was then discontinued as it was no longer necessary, and the absence of respiratory movement made the subsequent operative procedures easier.

In the first group of seventeen dogs, the right atrium was widely exposed by an incision extending from the right auricular appendage to the inferior vena cava parallel to the coronary sinus. The operative field was kept relatively bloodless by aspirating the cardiac venous blood. A large portion of the interatrial septum above and posterior to the coronary sinus was excised under direct vision. The defect was roughly circular. The extent of the opening was measured by calipers in two axes.

After creation of the defect most of the cardiac venous blood flowed into the left atrium by gravity. This reduced or eliminated the need for aspirating cardiac venous blood from the operative field. Physiologic saline solution was continuously injected into the left atrium in an attempt to displace air from the left atrium and so prevent fatal air embolism of the coronary arteries.

The previously fashioned pericardial graft was then placed over the defect and secured to the cephalad margin of the opening by two interrupted 00000 Deknatel silk sutures placed on each side of the graft (Fig. 2, *C*). The free inferior edge of the graft was easily manipulated by two previously placed sutures. The free extremity of the graft was then drawn upward and cephalically, and the cephalad margin of the defect sutured to the under surface of graft with a continuous suture. The graft was then replaced over the defect and two interrupted sutures were placed, one on each side of the graft, fixing it to the caudad margin of the defect. The graft was then fixed to the margins of the defect by three continuous sutures connecting the previously placed interrupted sutures (Fig. 2, *D*). In the first six experiments the pedicle was divided close to the cephalic suture line after the graft had been sutured to the margins of the septal defect. An attempt was then made to aspirate air trapped beneath the graft in the left atrium by means of a needle inserted through the suture line.

The incised wound in the wall of the right atrium was closed by a continuous suture. Aspiration of the cardiac venous blood was discontinued before complete closure so that the right atrium would fill with blood. Small pledgets of Gelfoam saturated with thrombin solution were sutured over the atrial wound. The ligatures about the venae cavae were divided thus returning a part of the total blood flow to the animal's heart and lungs. The lungs were insufflated with the respirator. The tubes leading to the extracorporeal circuit were clamped. The venous cannulae were removed and the azygos vein and right auricular appendage ligated. The arterial cannula was removed and the femoral artery ligated.

The thoracic wound was sutured in layers. The pleural cavity was drained by two catheters as long as serosanguineous fluid could be obtained, usually four hours. A dilute solution of protamine sulfate, 0.1 to 0.05 per cent, was slowly

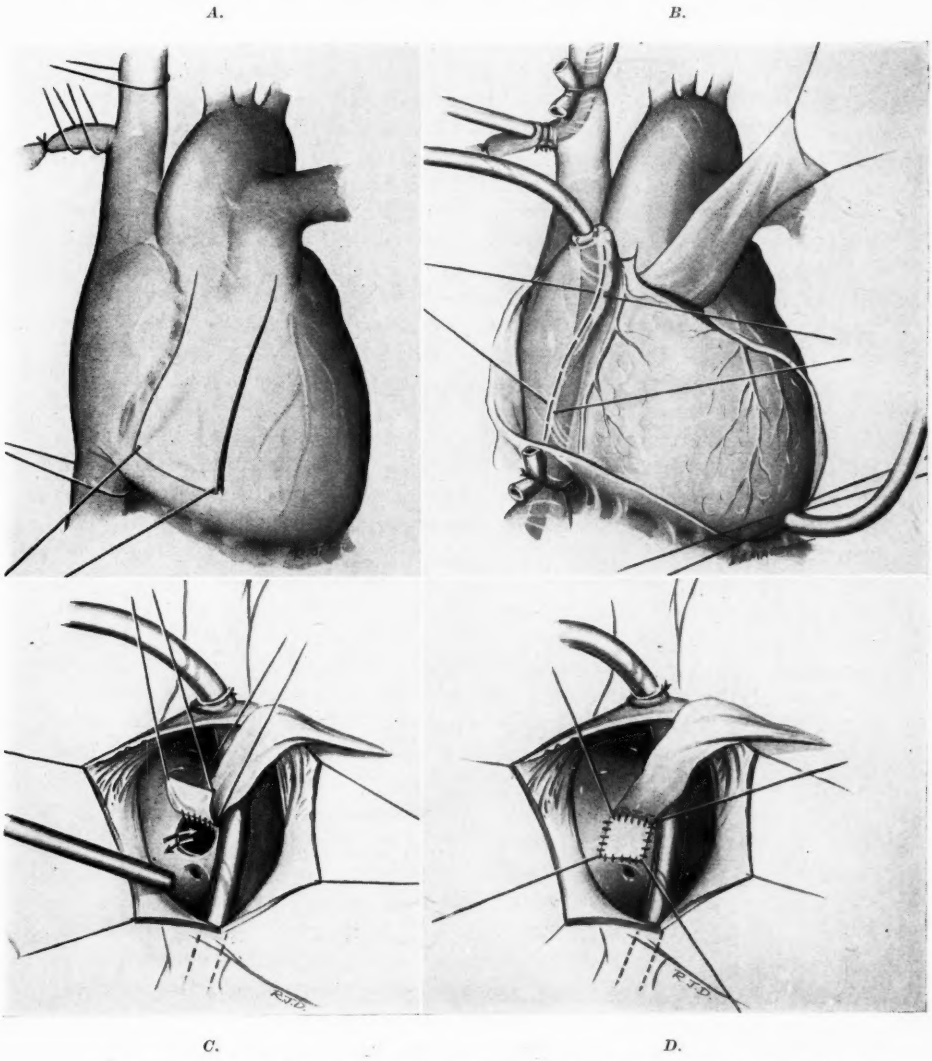


Fig. 2.—Method for closure of interatrial septal defects under direct vision with the assistance of an extracorporeal pump-oxygenator. *A*, Placement of ligatures around the venae cavae and azygos vein. Making the pericardial graft. *B*, The systemic venous return to the heart has been diverted by means of the cannulae placed within the venae cavae. The proposed line of incision in the right atrial wall is shown and the vent for the left ventricle has been established. *C*, The interatrial septum has been widely exposed and the sutures approximating the graft to the upper margin of the defect are shown. *D*, The graft has been completely sutured to the interatrial septal defect. The proposed line of division of the pedicle is shown.

TABLE I. PRODUCTION AND REPAIR OF INTERATRIAL SEPTAL DEFECTS WITHOUT A VENT IN LEFT VENTRICLE

EXP. NO.	DATE	WT. OF DOG KG.	TIME VENAE CAVAE OCCLUDED MIN.	TIME HEART OPEN MIN.	SIZE OF DEFECT CM.	BLOOD DETERMINATIONS DURING LATTER PART OF CAVAL OCCLUSION					INTERVAL BEFORE SACRIFICED (S) OR DEATH (D) DAYS	REMARKS
						OXYGEN SATURATION		ARTERIAL BLOOD pH	HEMATOCRIT PER CENT	HEMOLYSIS IN PLASMA MG. HB/100 ML.		
						VENOUS BLOOD PER CENT	ARTERIAL BLOOD PER CENT					
	1952											
0-497	Oct. 1	10	55	44		64	104	7.43	32	194	S—92	Pedicle graft
0-498	Oct. 3	11	60	42	1.5	42	75	7.33	30	174	S—90	Pedicle graft
0-499	Oct. 6	10	32	27	1.2 × 0.9	70	105	7.37	33	51	D	Died 4½ hr. from P.O. hemorrhage
0-501	Oct. 10	11	41	36	1.6 × 1.0	62	97	7.38	38	74	S—116	Pedicle graft
0-502	Oct. 14	11	57	50	1.5 × 1.2	60	99	7.30	43	49	S—157	Pedicle graft
0-503	Oct. 16	11	43	32	1.5 × 1.4	46	98	7.35	40	95	D	Died during procedure from air embolism
0-505	Oct. 24	12	37	33	1.9 × 1.5	59	98	----	38	216	S—147	
0-506	Oct. 28	12	70	22	--------	44	72	7.38	43	118	D	Died during procedure from air embolism
0-507	Oct. 30	11	36	29	1.5 × 1.2	53	81	----	37	51	S—141	
0-508	Nov. 3	10	37	34	--------	63	95	7.52	28	60	S—63	
0-520	Dec. 17	11	33	30	2 × 2	47	100	7.42	34	40	S—14	
0-521	Dec. 19	10	44	41	--------	41	95	7.37		89	D	Died in 24 hr. from overdose M.S.
0-522	Dec. 22	11	43	41	--------	62	104	7.35	32	54	S—22	
0-523	Dec. 29	13	31	35	2 × 2	62	102	7.42	38	96	D	Died during procedure from air embolism
	1953											
0-524	Jan. 6	12	39	35	2	61	103	7.47	34	87	S—7	Air in cor. circuit
0-526	Jan. 16	13	47	43	--------	70	103	7.30	43	65	D	Died in 10 hr. from P.O. hemorrhage
0-527	Jan. 16	13	40	41	1.5 × 2	64	103	7.35	38	65	S—17	Air in coronary circulation
Average		11	44	36		57	96	7.38	36	93		

injected intravenously until the coagulation time of the blood had approached a normal value. The animals received 300,000 units of procaine penicillin and 0.25 Gm. of streptomycin intramuscularly following the operation. Antibiotic therapy was continued for five to ten days postoperatively. All wounds healed per primam.

In the next group of seven dogs, a vent was established in the left ventricle to permit removal of air and blood during the production and repair of the interatrial septal defect. In two dogs a plastic cannula was inserted through the left ventricular wall in the angle between the circumflex and anterior descending branches of the left coronary artery. Blood and air escaped from this vent during the time the left atrium was open to the atmosphere. The blood accumulating in the pleural cavity from this vent was aspirated and returned to the extracorporeal circuit. This type of vent proved to be unsatisfactory and in the remaining five dogs a stab wound was made in the apex of the left ventricle and a small bore Tygon tube inserted into the chamber for a distance of approximately 3 cm. The tube was inserted into the left ventricle before the right atrium was opened, but after the venae cavae had been tied over the cannulae. The cardiac venous blood passing through the pulmonary circulation was thus removed from the left ventricle and diverted to the extracorporeal circuit. Once the vent had been established in the left ventricle, the right atrium could be opened and the interatrial defect created and repaired without danger of air embolism in the systemic arteries. While the communication between the two atria existed, bubbles of air could be seen passing through the Tygon tube leading from the left ventricle. After repairing the septal defect with the pericardial grafts, the right atrium was closed as described above. The ligature around the superior vena cava was then divided and the tube connecting the cannula in the superior vena cava to the extracorporeal circuit was clamped. The blood from the superior vena cava was returned to its natural course through the heart and lungs and washed out through the left ventricular vent any air trapped in the left side of the heart. When bubbles were no longer seen in the tubing leading from the left ventricle, the Tygon tube was removed and the stab wound in the apex of the left ventricle closed with interrupted sutures.

OPERATIVE RESULTS

A. Without Left Ventricular Vent.—Interatrial septal defects were produced and repaired by the technique just described in seventeen dogs without establishing a left ventricular vent to avoid air embolism. Eleven animals survived the perfusion and operative procedure. These animals were sacrificed between 7 and 157 days after operation (Table I).

In two instances (Dogs 524 and 527), air embolism of the coronary arteries occurred during the repair of the defect. The left coronary artery was completely filled with air and ventricular fibrillation occurred. Attempts at removal of the air by needle aspiration were unsuccessful. Consequently, the terminal portion of the anterior descending branch of the left coronary and marginal branch of the right coronary artery were divided near their points of termina-

TABLE II. PRODUCTION AND REPAIR OF INTERATRIAL SEPTAL DEFECTS WITH A VENT IN LEFT VENTRICLE

EXP. NO.	DATE	WT. OF DOG KG.	TIME VENAE CAVAE OCCLUDED MIN.	TIME HEART OPEN MIN.	SIZE OF DEFECT CM.	BLOOD DETERMINATIONS DURING LATTER PART OF CAVAL OCCLUSION					INTERVAL SACRIFICED (S) OR DEATH (D) DAYS	REMARKS
						OXYGEN SATURATION						
						VENOUS BLOOD PER CENT	ARTERIAL BLOOD PER CENT	ARTERIAL BLOOD pH	HEMATOCRIT PER CENT	HEMOLYSIS IN PLASMA MG. HB/100 ML.		
	1953											
0-529	Jan. 21	13	44	31	1.7 × 1.7	46	101	7.36	39	85	S—22	
0-530	Jan. 23	12	36	42	1.8 × 1.8	69	100	7.35	39	63	D	Died in 10 hr. from P.O. hemorrhage
0-537	Feb. 12	23	49	54	2 × 2	56	103	7.09	40	63	D	Died in 9 hr. from P.O. hemorrhage
0-538	Feb. 16	33	45	40	1.9 × 1.9	--	---	----	47	44	S—8	
0-539	Feb. 18	16	40	48	2 × 2	40	102	7.38	39	66	D	Died in 16 hr. from P.O. hemorrhage
0-540	Feb. 20	14	30	32	1.5 × 1.5	53	95	7.36	32	51	S—8	
0-543	Mar. 2	30	50	54	1.8 × 2	70	103	7.36	39	80	D	Died in 20 hr. from P.O. hemorrhage
Average		20	42	43		56	101	7.32	39	65		

tion and the coronary circulation permitted to pump itself free of air. The cut ends of the arteries were then ligated after the septal defect had been repaired and air could no longer be seen in the coronary arteries. Normal rhythm was established in Dog 524 by injection of 2 ml. of a 10 per cent solution of calcium chloride. A defibrillating shock was necessary in addition to calcium chloride in order to establish normal rhythm in Dog 527. Continuous massage was not necessary since the extracorporeal circuit maintained adequate systemic blood pressure and coronary circulation.

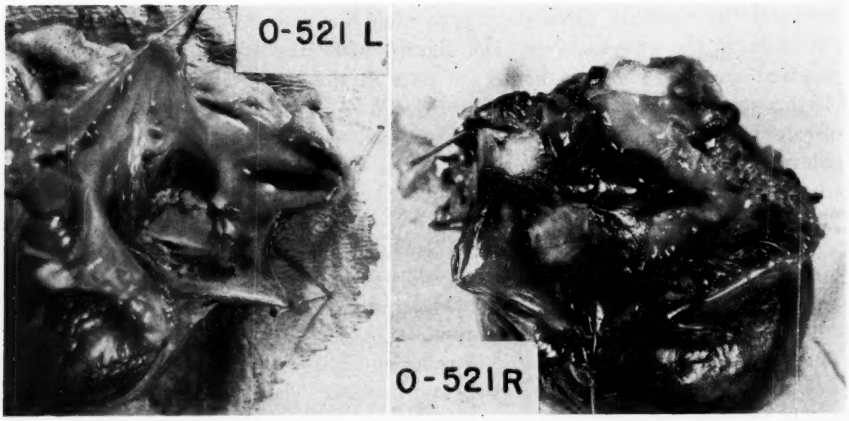
Dogs 499 and 526 died within ten hours after operation. Autopsy revealed large amounts of blood in the pleural cavities of both animals. Dogs 503, 506, and 523 died during the operative procedure from ventricular fibrillation resulting from air embolism of the coronary arteries. Dog 522 was alive twenty-four hours after perfusion. An intramuscular injection of morphine sulfate was given because the dog was restless and howling. The dog died fifteen minutes after the injection.

B. With Left Ventricular Vent.—A left ventricular vent was established before the production of the interatrial septal defect in seven dogs (Table II). In Dogs 529 and 530 the vent was placed in the angle between the circumflex and the anterior descending branches of the left coronary artery. In the remaining five dogs, a Tygon tube was placed within the left ventricle through a stab wound in the apex. Air embolism of the coronary arteries did not occur in any of these seven dogs. Four dogs died within twenty-four hours after operation. At autopsy a large amount of blood was found in the pleural cavities of all four dogs. Death was presumed to be due to uncompensated postoperative blood loss.

RESULTS AT AUTOPSY

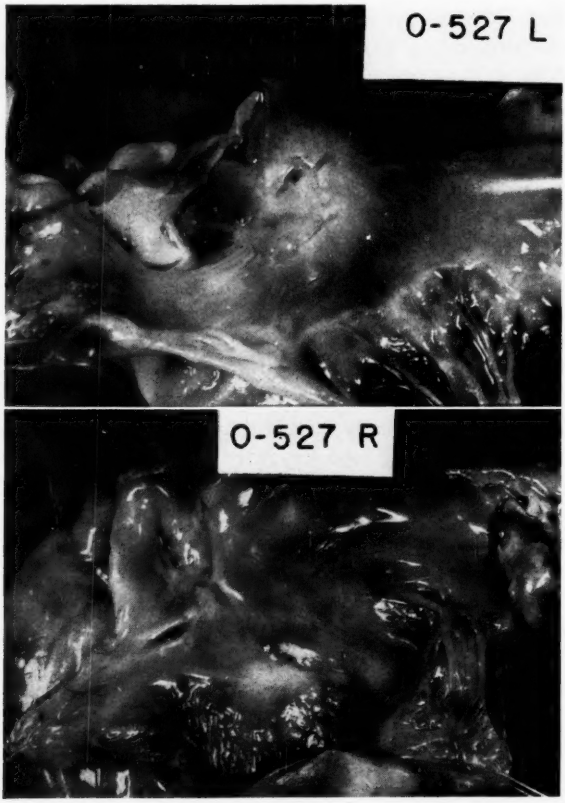
All surviving dogs were sacrificed. The septal defects were completely closed in all animals. Grafts observed within twenty-four hours following repair were covered with a thin layer of fibrin (Fig. 3, *A*). Three weeks after operation the graft was firmly united to the margins of the defect, and all surfaces and suture material were covered with smooth glistening endothelium (Fig. 4, *A* and *B*). Histologically at this time there was very slight inflammatory reaction and beginning fibroplasia (Fig. 4, *C*). At the third and fourth month healing was complete (Fig. 5, *A*, *B*, and *C*) and (Fig. 6, *A* and *B*). The pericardial pedicles in Dogs 497, 498, 501, and 502 were contracted fibrous cords which obviously contributed nothing to the vascular nutrition of the graft. None of the free grafts were necrotic and none failed to unite with the margins of the defect.

Interatrial septal defects were created, but not repaired, in five control animals. After establishing a vent in the left ventricle, the right atrium was opened and a portion of the interatrial septum excised. The defects were all greater than 1 cm. in at least one axis. The wound in the right atrium was then sutured. It was always necessary to re-establish complete blood flow through the heart before all air could be displaced from the left ventricle through the ventricular vent.



A. *B.*

Fig. 3.—Twenty-four hours after repair of interatrial septal defect with free graft. *A*, View from left atrium. *B*, View from right atrium.



A.

B.

Fig. 4.—Seventeen days after repair of interatrial septal defect with free pedicled graft. *A*, View from left atrium. *B*, View from right atrium.

Three of these animals survived the perfusion and operative procedure (Table III). Postoperatively, the soft systolic murmur which could be heard for a few days disappeared and no cardiac abnormality could be determined by auscultation. The animals were sacrificed at twenty-three, forty-four, and fifty-one days. The hearts were not enlarged. There were large, unhealed,

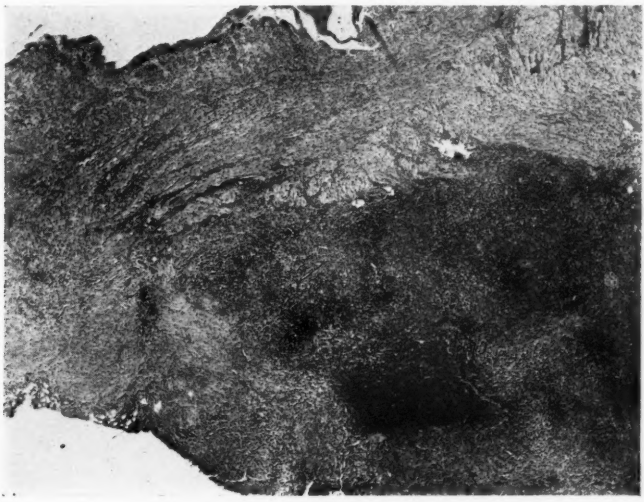


Fig. 4*C*.—Histologic appearance (×20).

interatrial septal defects in all three dogs. The defects were all approximately the same size as when they were made (Table III). The margins of the defects were smooth and glistening in appearance (Fig. 7, *A* and *B*).

In the twelve dogs in which a left ventricular vent was established there was no evident damage to the endothelium lining the left ventricle. The mitral valve leaflets, chordae tendineae, and papillary muscles within the left ventricle were all normal in appearance at death or sacrifice. No thrombi were present in the left ventricular chamber and there was no subendothelial ecchymosis or hemorrhage.

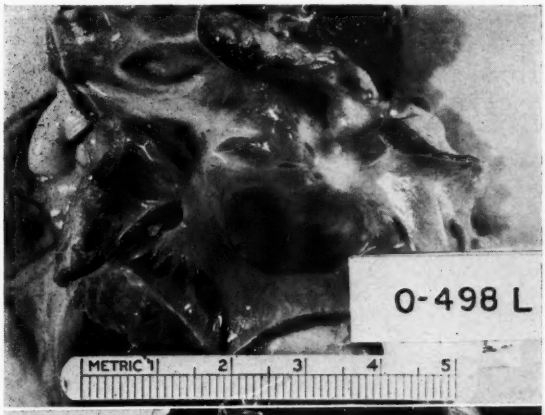
PERFORMANCE OF THE APPARATUS

In twenty-six of the twenty-nine experiments reported here, the saturation of arterial blood with oxygen was 95 per cent or more. The failure to obtain adequate oxygenation in three early experiments of the series (0-408, 506, and 507, Table I) is probably attributable to an error in technique during establishment of the blood film on the screens of the oxygenator.

The rate of blood flow through the extracorporeal circuit expressed in ml. per kilogram of body weight of the animal per minute was similar to that of previously reported series.[15] Only the rate of blood flow from the venae cavae was measured. It varied between 48 and 166 ml. per kilogram per minute, averaging 83 ml. per kilogram per minute. As we have previously pointed out, the degree of saturation of the venous blood with oxygen is prob-

TABLE III. CONTROL INTERATRIAL SEPTAL DEFECTS

EXP. NO.	DATE	WT. OF DOG KG.	TIME VENAE CAVAE OCCLUDED MIN.	TIME HEART OPEN MIN.	SIZE OF DEFECT CM.	OXYGEN SATURATION		ARTERIAL BLOOD pH	HEMATOCRIT PER CENT	HEMOLYSIS IN PLASMA MG. HB/100 ML.	INTERVAL BEFORE DEATH (D) OR SURVIVAL (S) DAYS	SIZE OF DEFECT AT TIME OF SACRIFICE	REMARKS
						VENOUS BLOOD PER CENT	ARTERIAL BLOOD PER CENT						
	1953												
0-531	Jan. 26	10.5	28	40	2.2 × 2.2	71	99	7.38	39	76	D		Died 36 hr., congested lungs
0-532	Jan. 28	11.5	23	20	1.6 × 1.8	47	98	7.36	37	32	S—51	1.4 × 1.2	
0-534	Feb. 2	12.0	22	19	1.5 × 1.5	70	103	7.47	42	49	D		Died 18 hr., congested lungs
0-535	Feb. 4	12.0	22	20	1.4 × 1.4	55	102	7.38	35	32	S—44	1.2 × 1.0	
0-541	Feb. 25	10.0	19	30	1.1 × 0.9	52	96	7.34	39	92	S—23	1.0 × 0.8	
Average			23	26		59	100	7.39	38	56			



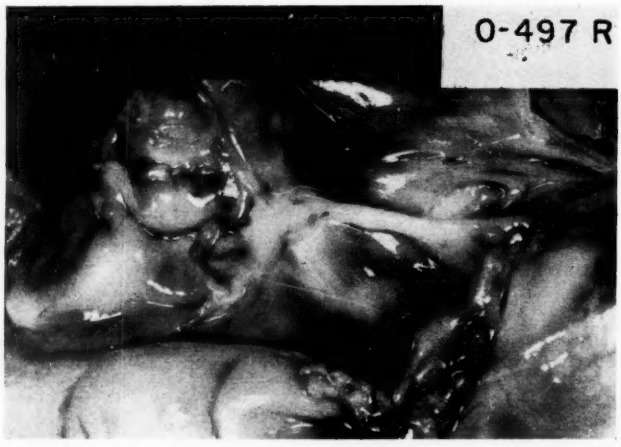
A.

B.

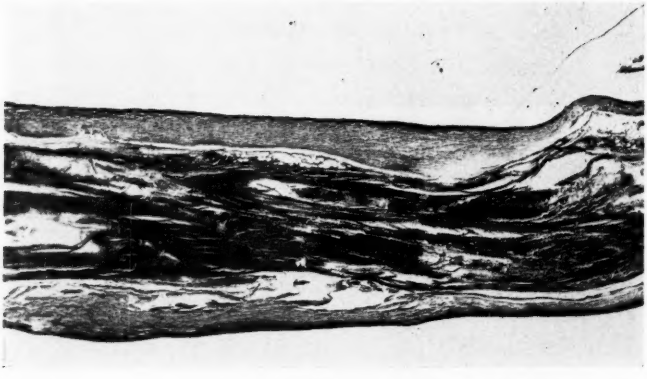
C.

Fig. 5.—Three months after repair of interatrial septal defect with pedicled graft. *A*, View from left atrium. *B*, View from right atrium. *C*, Histologic appearance (×20).

ably a better index of tissue oxygenation than the relation of blood flow to body weight. In these 29 experiments the venous oxygenation saturation was between 40 and 50 per cent in eight experiments. In two of these experiments the oxygenator was inadequately saturating the arterial blood with oxygen. In the



A.



B.

Fig. 6.—Four months after repair of interatrial septal defect with pedicled graft. *A*, View from right atrium. *B*, Histologic appearance (×20).

other six instances the flow rate was probably insufficient to maintain normal tissue oxygenation even though the arterial blood supplying the tissues was between 95 and 102 per cent saturated with oxygen.

With one exception (0-537, Table II) the pH of the arterial blood was maintained at 7.3 or above. As has been shown[14] there is a slight tendency for fixed acids in the blood to increase during the course of a perfusion. This increase on

the average amounts to only 3 mM per liter. To compensate for this increase, the carbon dioxide tension of the blood is automatically reduced thus maintaining the pH at a near normal level.

The amount of hemolysis occurring during these experiments is lower than in any series we have previously reported. In four of the early experiments in this series the hemolysis was above 100 mg. per cent free hemoglobin in the plasma. In all other experiments it was well below this level.

A. *B.*

Fig. 7.—Patent interatrial septal defects. *A*, Forty-four days after production. View from right atrium. *B*, Fifty-one days after production. View from right atrium.

SUMMARY

1. The cardiorespiratory functions of twenty-nine dogs have been maintained by an extracorporeal blood circuit while the right atrium was widely opened and an interatrial septal defect created under direct vision.

2. The septal defect was not repaired in five dogs. Three of these survived and were sacrificed at twenty-three, forty-four, and fifty-one days. There was no spontaneous closure or significant decrease in size of the defects.

3. In twenty-four of the twenty-nine dogs, after creating the septal defect, it was immediately closed by a pericardial graft. Fourteen of the twenty-four dogs survived and were sacrificed at varying intervals up to 157 days. In all instances the defect was completely closed, the graft well healed and covered with endothelium on both sides.

4. In twelve consecutive experiments a plastic tube vent was placed in the left ventricular chamber during the period in which the left atrium was exposed to atmospheric air. Air embolism of the coronary arteries did not occur in any of these experiments.

5. In the seventeen experiments in which the left atrium was exposed to atmospheric air in the absence of a left ventricular vent, air embolism of the coronary arteries occurred in five dogs. Three of the five dogs died.

6. Ventricular fibrillation occurred in the five dogs with coronary air embolism. It did not occur in any other animal.

The authors wish to acknowledge the assistance of Dr. Joseph McCloskey for histologic interpretation, and Miss Joanne Crothers, Dr. Maria Stegmanis, Dr. Alice Plume, and Miss Grace Small, for technical assistance.

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(For discussion, see page 631.)

AN OXYGENATOR-PUMP FOR USE IN TOTAL BY-PASS OF HEART AND LUNGS

Laboratory Evaluation and Clinical Use

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THE insistent challenge of surgical problems in cardiology and the potential application of corrective surgical measures have stimulated the search for mechanical substitution for the lungs and heart. Although this is a problem of immense complexity, the earlier progress by several investigators[1-4] has raised the hope that extracorporeal oxygenation and circulation can eventually be successfully employed in surgical cardiology. Results obtained through initial experiments with the Clark heart-lung apparatus have encouraged further investigations along these lines.

In experiments described in this report, it was not primarily planned to obtain long survival of the animal subjected to extracorporeal oxygenation and circulation. The first objective in this series was the continuation of laboratory evaluation of the Clark oxygenator-pump, with special reference to its efficiency in the processes of oxygenation and the elimination of carbon dioxide. The second objective was to acquire further experience in opening the cardiac chambers while the animal's functions of respiration and circulation are being carried out by this artificial means, that is, in the period that has been referred to as *the interval of total by-pass of the heart and lungs.* It was in the pursuit of this second objective that several new problems became apparent, and these unexpected developments necessitated a succession of modifications in the method of the experiment. More than that, these unexpected results had the fundamental effect of altering somewhat the hopeful anticipation of successful surgery in the widely opened and "bloodless" heart.

In the final part of this presentation a report is given of the clinical application of this machine. A complex defect in the cardiac septa was corrected by a direct operative procedure through a right auricular cardiotomy. A full description of problems associated with this case is given. It is believed that the publication of certain inherent difficulties involved will be of assistance to those who are engaged in surgical research in cardiology.

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This project was supported by grants from the Lucie Rawson Scientific Fund, the Cincinnati Children's Heart Association, the American Heart Association, the United States Public Health Service, and the Playtex Park Research Institute.

Read at the Thirty-third Annual Meeting of The American Association for Thoracic Surgery, San Francisco, Calif., March 27, 28, and 30, 1953.

*Chairman of the Department of Biochemistry of the Fels Institute for Research in Human Development.

METHOD

Many of the details of these experiments were exactly as described in an earlier publication,[5] and therefore only the significant innovations are given here. There was a much wider variation in body weight of the dogs. The lightest was 2.6 kilograms and the heaviest 23.2 kilograms. Pyribenzamine was included in the agents for premedication in every instance. Observations on body temperature were made by means of an esophageal thermometer, and the range was 27.8° C. to 32.5° C. during the period of total by-pass. Pentothal Sodium solution was used as the anesthetic agent in the latest studies. A polarograph[6] was employed to detect changes in the oxygen tension in various tissues, most frequently in the musculature of the neck and the thoracic wall.

The system of cannulation for the drainage of the systemic venous blood did not include the L-shaped cannula employed in earlier experiments. The superior vena cava was drained by means of a plastic tube with multiple perforations, which was passed down through the right jugular vein. The inferior vena cava was drained by two similar catheters with multiple perforations. These latter instruments were inserted and passed upward through the femoral veins.[7] Complete occlusion of the venae cavae was obtained by ligation of these vessels at the level of their junction with the right auricle.

Heparin was the only anticoagulant employed, and protamine was given at the conclusion of each experiment. The dose of the anticoagulant was determined by calculation from the estimated blood volume of the subject animal and the volume of blood required to fill the extracorporeal system; the calculated amount was 20 mg. of heparin per 1,000 ml. of blood. A simple protamine titration was carried out to determine the amount of this agent necessary to reverse the heparin effect at the conclusion of extracorporeal circulation.

Both femoral arteries were cannulated for the return of arterialized blood into the animal from the machine. Filters employing 325 mesh stainless steel screens, with a baked silicone coating, were inserted in the arterial line between the pumping chamber and the animal. (The filters were not used in every experiment since their effectiveness has not been fully established.) The system of cannulation for the withdrawal of the coronary venous return to the right half of the heart underwent some evolution during the study. The exposure gained through wide opening of the right auricle made cannulation of the coronary sinus a simple matter. However, there was often a relatively large volume of blood that returned via the several anterior cardiac veins,[8] and, to deal with the problem raised thereby, it was necessary to alter the system. This was accomplished by the development of a suction tube with a rounded, closed end and a single lateral opening near the tip. (Even if the lateral opening is occluded by an endocardial surface, the suction applied through this tube may never exceed 10 cm. of water.) This suction instrument was inserted through the tricuspid valve after the auricle had been opened. The application of suction in the chamber of the right ventricle

accomplished more complete removal of the blood from the coronary system. That is to say, coronary blood returning to the right auricle gravitated through the open tricuspid valve into the ventricle, and the additional fraction of the coronary venous system drained by the Thebesian vessels into that chamber was also withdrawn by the aspirator. The complete unit for the collection of coronary venous blood was attached so that it served as a tributary to the inlet (venous) side of the extracorporeal circuit.

OBSERVATIONS ON OXYGENATION AND ELIMINATION OF CARBON DIOXIDE

There was a rapid reduction in body temperature of the dogs in the first quarter hour of "total" extracorporeal circulation. Observations on the oxygen content of samples of arterial and venous blood must be carefully interpreted with consideration of the existing hypothermic state.

The output of the mechanical heart, that is, the flow rate, is given in Table I. No difficulty was encountered in draining the venae cavae, and a free flow of venous blood was obtained away from the dog and into the extracorporeal circuit. (An increase in the suction required for venous pickup was invariably an early sign of the need for a small blood transfusion.) The criteria considered important in the control of this aspect of the experiments were the maintenance of the mean arterial pressure within a physiologic range and continual oxygenation which would ensure at least 90 per cent saturation of the stream returning from the apparatus into the animal. Therefore, when these criteria were satisfied, no additional effort was considered necessary to attain an arbitrary value for flow rate (cubic centimeters per minute per kilogram).

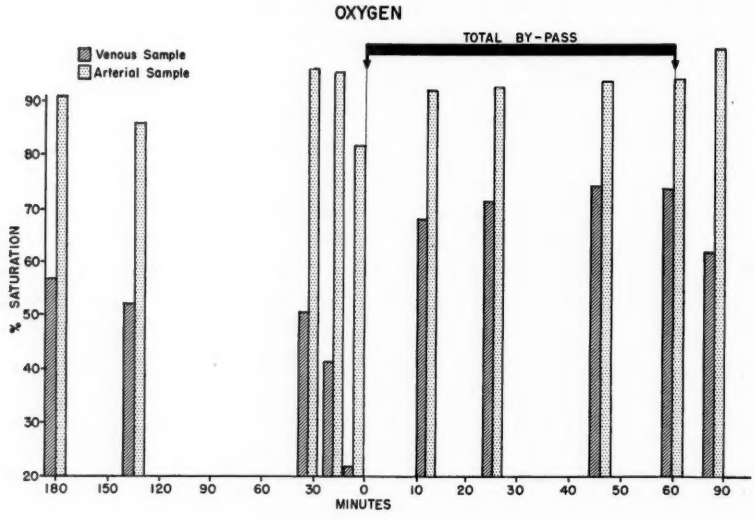
The determination of oxygen and carbon dioxide in blood samples was carried out in 18 experiments. The venae cavae were completely obstructed for sixty minutes and arterial and venous blood samples were drawn during this hour, as well as before and after it. The oxygenation that was accomplished in the extracorporeal apparatus is shown graphically in Fig. 1. From this it was apparent that there was no marked change during the hour of artificial oxygenation in the oxygen saturation of arterial samples from the animals. The per cent saturation remained in the range of 90, which was the approximate level of control values. Additional pertinent data are listed in Table I. The animal's body temperature and corresponding machine flow rates are correlative factors. An expression of oxygenation is given by the mean value of 3.67 c.c. per minute per kilogram of body weight.

The elimination of carbon dioxide in the extracorporeal circuit is shown in Fig. 2. The carbon dioxide content of arterial and venous samples varied somewhat in the pre-by-pass controls. There was no accumulation of carbon dioxide during the one-hour interval of caval occlusion. (Supplementary data are given in Table I.) Another measure of the elimination of carbon dioxide is the mean value of 2.99 c.c. per minute per kilogram of animal weight.

CARDIOTOMY (RIGHT AND LEFT AURICLE) DURING "COMPLETE" BY-PASS

The oxygenator-pump which was found to be an efficient mechanical substitute for the lungs and heart in laboratory experiments has made visual exploration of the cardiac chambers of dogs a feasible procedure. A long incision

Fig. 1.



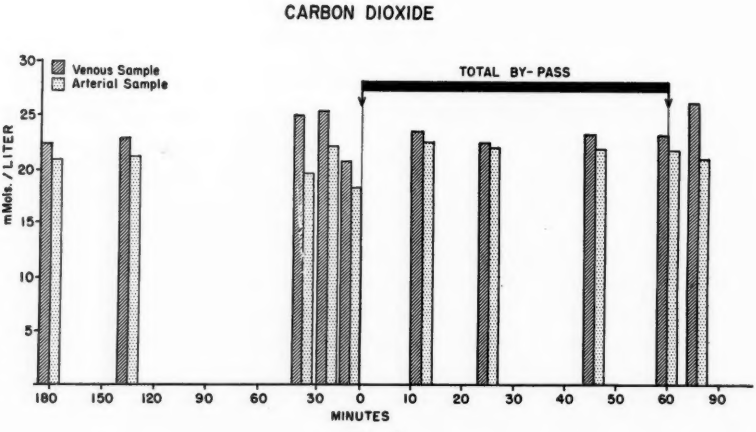


Fig. 2.

Fig. 1.—Oxygen saturation of arterial and venous samples during total by-pass. Each column represents an average of 3 to 5 analyses of samples drawn before and following the total by-pass. The columns in the interval of by-pass represent an average of from 5 to 12 analyses. The figure records the analyses of 105 samples (52 arterial and 53 venous).

Fig. 2.—Carbon dioxide content of arterial and venous samples during total by-pass. Each column represents an average of 3 to 5 analyses of samples drawn before and following the total by-pass. The columns in the interval of by-pass represent an average of 5 to 14 analyses. The figure records the analyses of 104 samples (52 arterial and 52 venous).

TABLE I. FLOW RATES, OXYGENATION AND CARBON DIOXIDE DETERMINATION

WEIGHT OF DOG (KG.)	BODY TEMPERATURE (°CENTIGRADE)	TIME AFTER START OF TOTAL BY-PASS (MINUTES)	FLOW (C.C. PER MINUTE PER KG.)	OXYGENATION (C.C. PER MINUTE PER KG.)	CO_2 ELIMINATION (C.C. PER MINUTE PER KG.)
11.4	27.8	9	79.8	1.84	2.39
	27.5	27	91.2	3.47	0.18
14.5	30.3	15	98.6	3.35	1.48
	30.5	45	98.6	2.47	1.97
13.2	31.0	29	98.5	2.56	2.46
8.6	28.1	35	97.7		3.52
16.0	32.0	25	97.5	3.61	3.9
	31.5	45	87.5	4.64	4.55
12.0	31.2	41	105.0	3.47	5.15
10.0	31.0	44	90.0	3.06	2.97
11.1	32.4	17	118.9	4.99	1.66
	32.0	35	108.1	3.24	4.54
	31.2	56	118.9	3.57	3.57
23.2	29.7	48	103.4	3.62	1.76
	29.5	55	103.4	3.00	4.14
10.0	29.0	61	144.0	4.46	3.31
16.3	31.4	15	88.3	3.71	3.62
	30.8	10	88.3	4.15	3.71
11.4	29.6	48	126.3	3.16	1.52
15.0	32.5	14	86.7	4.77	3.73
	32.0	39	91.0	5.19	3.37
14.1	29.8	45	92.2	1.94	3.50
	29.6	59	92.4	2.30	2.12
3.9	28.2	35	135.9	5.03	4.21
3.39	28.0	33	129.8	4.80	2.60
2.6	31.1	11	84.6	5.33	1.78
Mean			102.2	3.67	2.99

extending from the base of the right auricular appendage to the insertion of the inferior vena cava was made in 8 dogs. With total occlusion of both cavae, the lowest portion of the right auricle was still usually flooded by the volume of venous return from the coronary sinus, from the anterior cardiac veins, and from other coronary venous channels draining into that chamber. The volume of this fraction of the coronary venous return was estimated in several experiments. The results given in Table II do not represent an accurate measurement of coronary blood flow; they serve only as a quantitative expression of this characteristic of the opened and by-passed heart, an aspect not sufficiently emphasized heretofore. In every instance, this volume of coronary venous blood was large enough to disappoint greatly the surgeon who had been expecting to operate in a "bloodless" field within the heart.

Incision of the wall of the right auricle did not alter the contractions of the left auricle or the ventricles. The blood which drained from the coronary sinus often shot into the lower part of this auricular chamber in jets synchronous with ventricular systole. This fraction, together with the other portions of coronary venous return, rapidly coursed over the lower rim of the tricuspid orifice into the right ventricle. The stream of blood gravitating down the dependent portion of the auricle and over the tricuspid rim was necessarily turbulent. This activity caused the mixture of some air with the blood, to

form bubbles which were swept along into the right ventricle. It was clear that this hazard of bubbles in the right ventricle accompanied every extensive right auricular cardiotomy.

TABLE II. VOLUME OF CORONARY VENOUS BLOOD DRAINED FROM OPENED HEART DURING "TOTAL BY-PASS"

WEIGHT OF DOG (KG.)	MEAN ARTERIAL PRESSURE (MM. HG)	BODY TEMPERATURE (°CENTIGRADE)	ARTERIAL O_2 (% SATURATION)	PULMONARY ARTERY	AORTA	AURICULAR SEPTUM	MAXIMUM VOLUME CORONARY VENOUS BLOOD ASPIRATED (% TOTAL MACHINE OUTPUT)
Aspiration of Coronary Sinus							
8.6	85	28.6	100	Open	Open	Intact	10 (1.19%)
13.2	95 to 115	28.1		Open	Open	Intact	25 (2.66%)
Aspiration of Dependent Portion of Right Auricle							
12.0	80 to 120	31.5	95.9	Open	Open	Intact	60 (4.76%)
10.0	95 to 105	32.0	100.0	Open	Open	Intact	25 (2.5 %)
14.3	80	32.5		Open	Open	Open	12.5 (1.3 %)
13.0	65 to 70	31.8		Open	Open	Intact	40 (3.3 %)
	73 to 75	31.1		Closed	Closed	Intact	7 (0.58%)
	70 to 75	30.7		Open	Open	Intact	240* (18.1 %)
Aspiration of Right Ventricle							
23.2	60 to 120	30.1	91.8	Open	Open	Intact	333 (13.9 %)
10.0	80 to 94	30.3	94.0	Open	Open	Intact	73 (5.0 %)
16.3	65 to 80	31.6	94.0	Open	Open	Intact	200 (13.9 %)
	80	30.6	93.8	Open	Open	Intact	280* (20.0 %)
11.4	70 to 100	29.6	95.0	Open	Open	Intact	190 (13.2 %)
15.0	40 to 100	32.5	77.5	Open	Open	Intact	170 (13.1 %)
14.1	80 to 150	30.0	81.1	Open	Open	Intact	140 (10.8 %)
2.6	75 to 80	30.1	108.8	Closed	Closed	Intact	26 (10.3 %)
4.9	100	29.8		Closed	Closed	Open	40 (13.3 %)

*Measurements taken during ventricular fibrillation.

The technical problems were increased still further by excision of the interauricular septum. This was so because, following the excision, the turbulent flow of blood and resultant air embolism involved the left half of the heart as well. The entire interauricular septum was removed and the two auricles were converted into a single, wide-open chamber in 8 dogs. The coronary venous inflow of the right auricle was admixed immediately with the richly oxygenated blood of the left auricle. The contents of the left auricle consisted of venous drainage from the bronchial vascular system, since the pulmonary artery flow had been interrupted and there was no patent ductus. Fractional measurements were not possible, but the volume of bronchial arterial flow which returned via the pulmonary veins was surprisingly large and could not be disregarded. However, this increment of "venous blood" to be drained away from the "totally by-passed" heart was the lesser of the complications which followed excision of the auricular septum. The blood in the active left auricle was soon mixed with air, just as had occurred in the right auricle, and it was but a short time until this air-blood mixture had coursed through the mitral valve and out into the aorta.

This description indicates the two principal problems encountered upon opening the right or both auricles. The first was the efficient drainage of the

venous blood of the intrinsic (coronary) cardiac circulation. The second was the admixture of air and this intrinsic venous blood with resultant air embolization. The following steps were taken successively in an effort to solve these problems. The first problem, the excessive volume of coronary venous return to the right side of the heart, was dealt with initially by catheter drainage of the coronary sinus. In several animals, however, the coronary return through vessels *other* than the coronary sinus comprised so large a volume that the auricle was inadequately emptied by simple coronary sinus drainage.[9, 10] The next method that was tried for emptying the right auricle was continuous suction in the dependent portion of the chamber. At that stage in the evolution of the procedure, the hazard of air embolism became more apparent and a further modification in method was tried. This was to maintain continuous suction in the right ventricle by means of one of several different glass and plastic catheters, which served to withdraw the coronary venous blood. It also prevented the propulsion of air emboli into the pulmonary artery, since the pressure in the right ventricle was reduced by continuous suction and the instrument separated the leaves of the tricuspid valve. The "blind" cannulation of the chamber through the tricuspid was somewhat hazardous because of the possibility of trauma to the endocardium. In addition to this risk, it was soon noted that no matter what aspirating device was employed for that purpose, it added to the complexity of a restricted operative field.

An alternate procedure which lacked these disadvantages was temporary occlusion of the pulmonary artery. This vessel was occluded by means of a ligature just beyond the pulmonic valve. After occlusion, an incision was made before the auricle became overdistended with coronary venous return. Satisfactory emptying resulted after the aspirator was placed in the dependent portion of the auricle. There was some regurgitation through the tricuspid valve during the period of pulmonary artery occlusion, but embolism of air into that vessel was effectively prevented.

As has been mentioned, removal of the interauricular septum exposed the major circulation to the hazard of air embolism. This complication was not prevented by continuous aspiration of the bronchial and coronary venous blood from the dependent portion of the left auricle. Frequently a mixture of air and blood passed the mitral valve and soon appeared in the coronary arteries. After failure of this first measure, the next method tried was that of total occlusion of the aorta. Obstruction of the aorta at the lowest level of its extracardiac portion prevented propulsion of air emboli into the major circulation. Closure of the aorta, however, interrupted the filling of the coronary arteries from the arterial stream of the extracorporeal circuit. It is true that in some dogs the coronary circulation could be maintained, despite interruption of flow along the ascending aorta, if the bronchial system of arteries and veins were to return an adequate volume of blood to the left half of the heart. In several dogs in the present series, however, this bronchial circuit did not supply a volume adequate for coronary filling, and complete closure of the aorta produced serious changes in the color and tone of the myocardium after only two to three minutes.

Failure of this second method led to the trial of a third precaution against the development of air embolism. The aorta was catheterized with an additional plastic tube of small diameter. The tip of this instrument was localized just above the aortic valve, thus serving as a special channel for the return of arterialized blood from the apparatus to the proximity of the coronary orifices. An adequate flow of arterial blood into the coronary circuit was thus assured, and the aorta was then occluded in its ascending portion by ligation and compression of its walls around the central catheter. The mixture of air and blood which passed through the mitral valve from the opened auricles was rapidly ejected by the ventricle, and shortly thereafter air embolization of the coronary arteries was again apparent. This method had maintained coronary artery filling by means of the separate axial catheter from the machine. It failed, however, to prevent the ventricle from ejecting blood, with air admixed, into the occluded root of the aorta. Thus the problem of air embolism remains unsolved; but there is no reason to accept it as an unpreventable hazard, inasmuch as several obvious methods for the prevention of air embolism have not yet been given adequate trial.

The drainage of blood from the open cardiac chambers may be accomplished with less difficulty because of the unique capacities of the Clark apparatus. These are the coalescence of bubbles and the continuation of pumping without interruption, despite the entrance of rapidly varying volumes of blood into the venous side of the circuit. These qualities of the machine make it appear now that the withdrawal of a large volume of blood from the opened (and totally by-passed) heart can readily be accomplished. What is more important, this volume of blood from the intrinsic (coronary) and bronchial circulations can be channeled directly into the extracorporeal circuit, so that the effect of an even brief reduction in the volume of extrinsic circulation is entirely prevented.

CLINICAL USE OF THE OXYGENATOR-PUMP

The patient, a 4-year-old white boy, had been under medical observation most of his life. Several weeks after birth, a bulging deformity was noted in the precordial area. In his first year there were severe infections of the respiratory tract, one of them developing into bronchopneumonia which necessitated hospitalization. There were similar episodes in the patient's second and third years, and during that period one of the infections was complicated by congestive heart failure. Following that, he continued on 0.5 mg. of digitoxin daily; his exercise tolerance became so poor, however, that in his fourth year he could walk only about 25 yards. He slept poorly because of orthopnea and a persistent, nonproductive cough.

The boy's weight was 12 kilograms, his height 95 cm., and he appeared to have been chronically ill. His jugular venous pressure was elevated to 5 cm. above the sternal angle; and the liver edge, which did not pulsate, was 6 cm. below the right costal margin. Blood pressure was 94/50 mm. Hg in both arms and 110/60 in both legs, and the pulsation of the femoral arteries was normal.

The precordial area bulged markedly and the apex was in the sixth left intercostal space in the anterior axillary line. A coarse systolic thrill was palpable down the left sternal border and a grade 4 systolic murmur was audible over the precordium and the back. The second sound was widely split, but there was no diastolic murmur.

Examination of the peripheral blood, urine, and erythrocyte sedimentation rates gave normal values. The electrocardiogram revealed incomplete right bundle branch block. Chest roentgenograms showed cardiac enlargement and pulmonary overcirculation. The total blood volume was estimated to be 990 c.c.

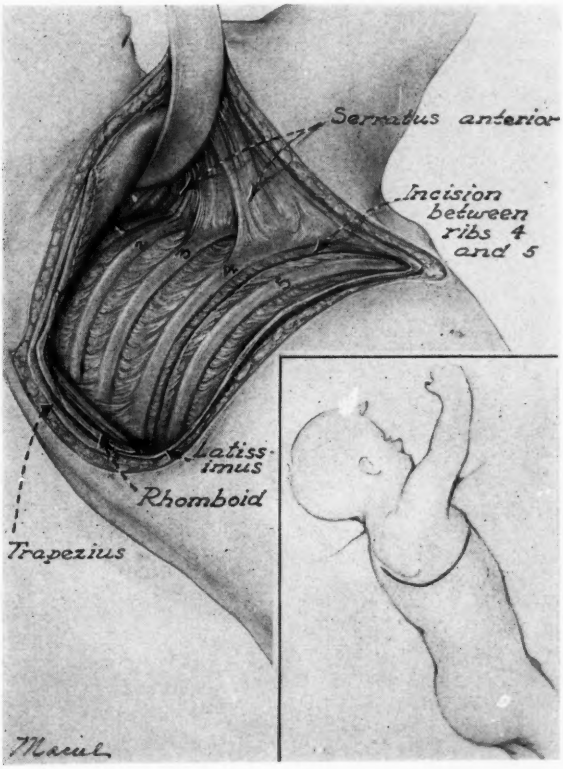


Fig. 3.—Position of patient on table, and location of posterolateral incision.

Cardiac catheterization five months prior to surgery gave the following results:

	Pressure	*O_2 content*
Pulmonary artery	92/38 mm. Hg	11.8 vol. %
Right ventricle	92/10	11.7 vol. %
Right auricle (mean)	10	11.9 vol. %
Superior vena cava	—	10.0 vol. %

Peripheral arterial oxygen saturation was 97 per cent; the cardiac output, 5.8 liters per minute; the calculated left-to-right shunt, 3.1 liters per minute. The working diagnosis was a defect in the interauricular septum, and surgical closure was planned because of the precarious condition of the child.

The patient was anesthetized with Pentothal Sodium solution, following which oxygen and ether were given through a closed endotracheal system. A moderate cooling effect was obtained by applying bags of cracked ice, and the operation was begun after the rectal temperature had been reduced to 90° F. The incision employed and the initial

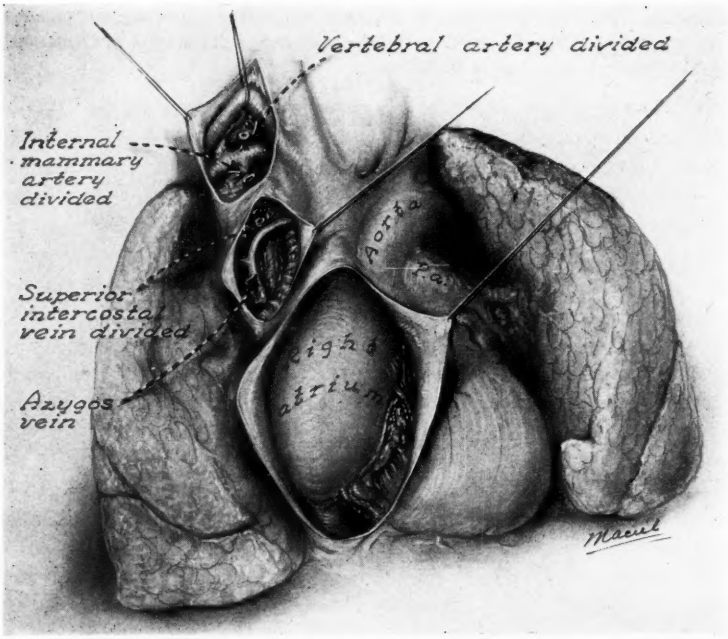


Fig. 4.—Exposure of right atrium; the subclavian artery and azygos vein for catheterization.

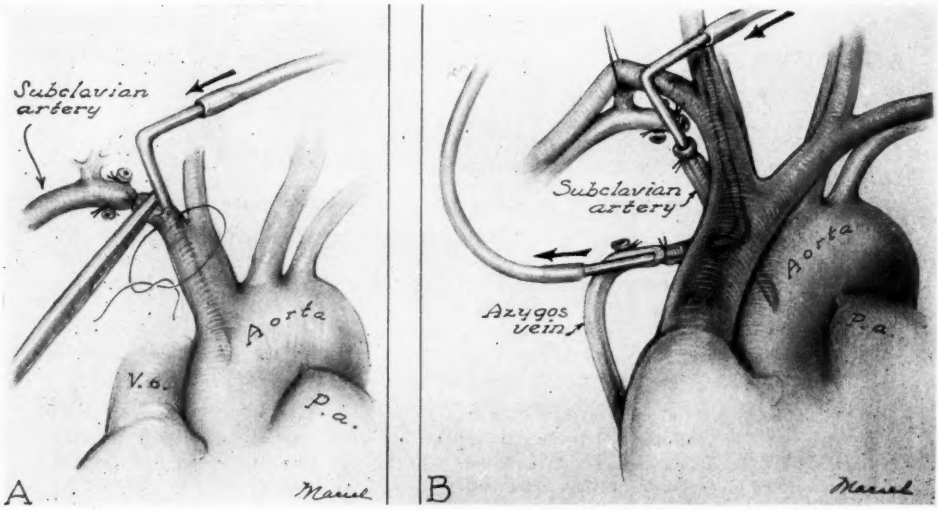


Fig. 5.—*A*, Cannula for return of blood from extracorporeal circuit into aorta. *B*, Catheter for drainage of the superior vena cava.

steps taken in the dissection within the chest are shown in Figs. 3 and 4. The catheter system for venous drainage included three plastic tubes with multiple perforations. Two of these were inserted via the saphenous veins into the inferior vena cava, their tips lying near the level of the renal veins. The other catheter was then passed through the

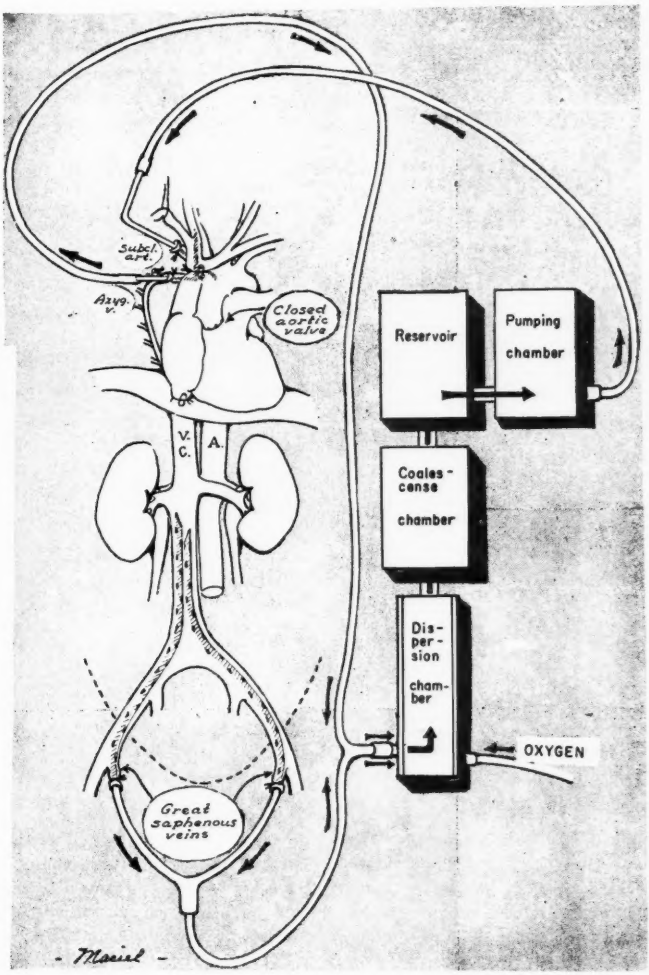


Fig. 6.—Oxygenator-pump showing attached venous pickup catheters and channel for return of blood into aorta.

azygos vein and upward along the full length of the superior vena cava. The channel for return of arterialized blood was a single plastic cannula, which was inserted through the subclavian artery and advanced until its tip was in the ascending aorta (Fig. 5).

The entire extracorporeal system was filled with blood, and care was taken to ensure complete replacement of all that had been lost up to that stage of the operation.

Three units of decalcified blood* (approximately 1,200 c.c.) and one unit of blood collected in the standard manner with ACD† were used for this. Fig. 6 gives in diagrammatic form the complete extracorporeal circuit and its attachments to the vena cavae and the aorta.

Extracorporeal circulation was started and the flow rate slowly accelerated, so that after ten minutes the rate was 900 to 1,200 c.c. per minute. The superior and inferior venae cavae were then occluded, and the wall of the right auricle was opened through a long incision. This chamber had seemed quite collapsed, as if it were empty, but within a few minutes after entering it the entire area was flooded by the great volume of returning coronary venous blood. The catheter system provided for the withdrawal of this coronary outflow was not adequate to handle the volume encountered. This insufficiency

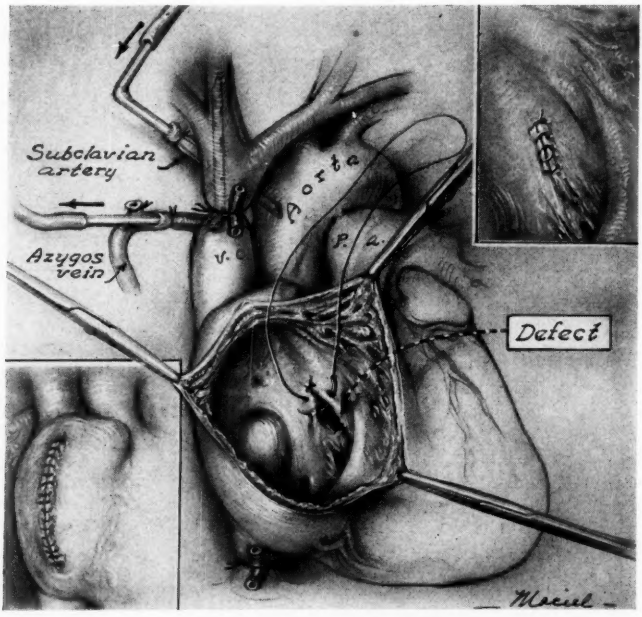


Fig. 7.—Exposure of septal defect through right auricular cardiotomy in by-passed heart.

resulted in a delay in returning the venous blood to the extracorporeal circuit and, as a result, the arterial pressure fell to shock levels. The inadequate system for draining away all the intrinsic blood was compensated for, in part, by several improvisations, but an uninterrupted and adequate machine flow could not be maintained. The volume of coronary blood aspirated from the right auricle was not less than 250 c.c. per minute. The arterial pressure did not return to a satisfactory level until approximately fifty minutes after the onset of shock.

The opening in the right auricle made it possible to close the defect by sutures placed under direct vision. The exposure that was gained and the simple method of closure are shown in Fig. 7. The complex anatomical nature of the defect and its effect upon the dynamics of the ventricles and the right auricle may be seen in Fig. 8. Despite the difficulty with an overwhelming coronary venous outflow, it was necessary to hold

*Fenwal Ion Exchange Blood Pack, Fenwal Laboratories, Inc., Framingham, Mass.
†Baxter Transfuso Vac Container, Baxter Laboratories, Inc., Morton Grove, Ill.

the auricle open for only seventeen minutes. The machine flow was 1,440 c.c. per minute after closure of the auricular wall, and the by-pass was terminated after a total of thirty-three minutes.

Protamine solution was then given intravenously and all catheters and cannulae were withdrawn. The pleural space was drained and the chest wall was closed in the standard manner. A dilute solution of Neosynephrine was given intravenously, as well as a transfusion of 250 c.c. of blood. Eight hours following operation the child was able to move his extremities and take sips of water. Sponging was required to prevent serious hyperthermia. He remained anuric and sixteen hours after closure of the defect he died.

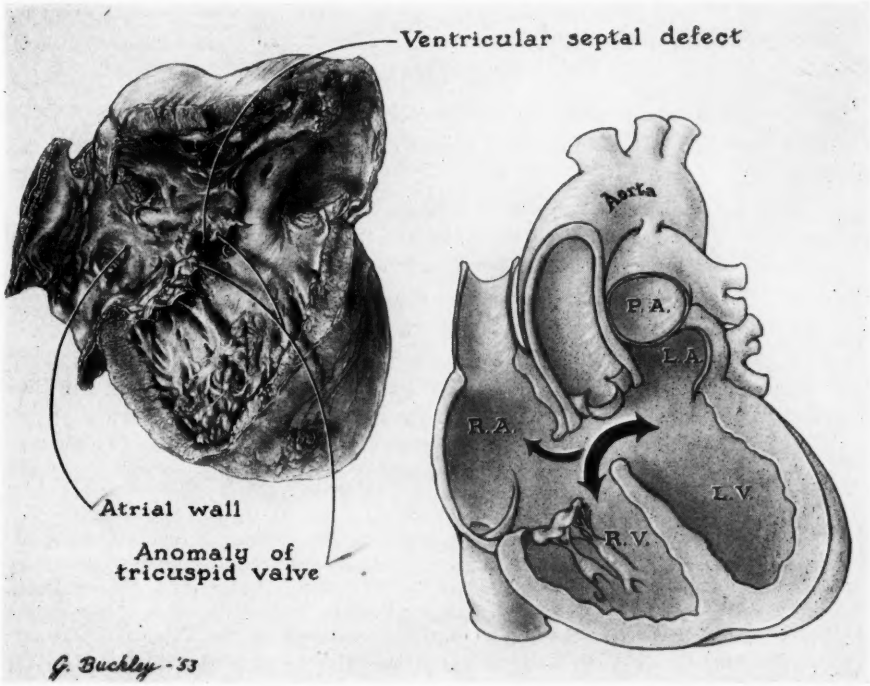


Fig. 8.—Ventricular septal defect and anomaly of tricuspid valve as viewed through opened right auricle and ventricle.

There was a small amount (90 c.c.) of blood in the right pleural space and some atelectasis in the dorsal portions of both lungs. The ventricular septal defect (1.5 cm. in diameter) had been closed by the surgical procedure. After removal of all sutures, it was shown that the right auricle communicated directly with the cavity of the left ventricle, because of the incomplete septal cusp of the tricuspid valve. There was no defect in the septum between the auricles. A fresh infarct was found in the upper pole of the right kidney. The brain was not examined, and therefore the possibility of cerebral air embolism was not expected.

The many observations made during this procedure could not all be included in this presentation, but the following summary covers the most important features. The preoperative diagnosis was not confirmed, but rather a defect high in the ventricular septum and an anomaly causing marked incompetence of the tricuspid valve were encountered after the by-passed heart was opened. A massive flood was caused by the coronary

venous return. The oxygenator-pump functioned perfectly, despite irregularity in the flow into its venous side, and despite the aspiration of very great amounts of air along with the blood from the intrinsic cardiac circulation. The explanation for the large volume of coronary venous return is a matter of fundamental importance. It is certain that a high degree of arterial oxygen saturation was present at the time the cardiotomy was started. The cerebrum, and possibly the myocardium as well, suffered hypoxia in the period of hypotension caused by the temporary reduction of machine flow rate. After suture of the septal defect and termination of the extracorporeal circulation, the heart resumed the emptying of its filled chambers and continued to beat normally. Death was caused by cessation of function of the higher centers. No sign of air embolism was found at the limited necropsy, and blood cultures taken at the conclusion of extracorporeal circulation proved to be sterile.

SUMMARY

1. Some innovations in the experimental method were discussed.

2. The machine flow rates and animal temperatures in 17 experiments were tabulated.

3. Results given of analysis showed that oxygenation was obtained at the rate of 3.67 c.c. per minute per kilogram, and that carbon dioxide was eliminated at the rate of 2.99 c.c. per minute per kilogram.

4. Experiences in dealing with the coronary venous return and the hazard of air embolization in the opened and "by-passed" heart were presented.

5. A description was given of an operation on a child with a defect in the ventricular septum. In the surgical procedure his heart was by-passed and the defect was closed by a direct approach through the opened right auricle. The problem created by excessive coronary venous return was discussed. The child's death, sixteen hours later, came as the result of a period of hypotension during the operation or cerebral air embolism.

The authors wish to express their gratitude to Drs. Thomas Largen and Frederic N. Silverman for their assistance with many problems encountered over a long period of time. The technical assistance contributed by Mrs. Molly Kaplan, Miss Ruth Parker, Miss Zena Taylor, and Miss Carol Limbert was essential to the completion of this work. Student anesthetists from the Department of Anesthesia of the Cincinnati General Hospital, under the direction of Miss Mary Alice Costello, were most helpful in carrying out the experiments.

The Vernet Equipment Foundation of Antioch College made available several valuable pieces of equipment for use in the laboratory.

The Dow-Corning Corporation of Midland, Mich., and the Corning Glass Works, Corning, N. Y., have continued in their interest and material assistance with technical problems.

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DISCUSSION ON "THE PRODUCTION AND REPAIR OF INTERATRIAL SEPTAL DEFECTS UNDER DIRECT VISION WITH THE ASSISTANCE OF AN EXTRACORPOREAL PUMP-OXYGENATOR CIRCUIT," BY DR. BERNARD J. MILLER, DR. JOHN H. GIBBON, JR., DR. VICTOR F. GRECO, DR. BURGESS A. SMITH, DR. C. HAROLD COHN, AND DR. FRANK F. ALLBRITTEN, JR.; AND "AN OXYGENATOR-PUMP FOR USE IN TOTAL BY-PASS OF HEART AND LUNGS," BY DR. JAMES A. HELMSWORTH, DR. LELAND C. CLARK, JR., DR. SAMUEL KAPLAN, AND DR. ROGER T. SHERMAN

DR. CLARENCE DENNIS, Brooklyn.—In 1947, work was started by a group of us at the University of Minnesota in an effort to develop a heart-lung apparatus of the type you have heard about today. Our original orientation was provided by Dr. Gibbon and his group in Philadelphia; we have enjoyed his friendship and exchange of data since that time, I think to the benefit of both groups, but I know to the benefit of ours. It is my opinion that Dr. Gibbon is not only the pioneer in this field, but that he unquestionably is the leader at the present time as well.

Initially we considered pumps to shunt blood around one side of the heart alone, but clinical indications for such applications seemed to us to be rare, and the propriety of application in those cases in which pumps have been used clinically is a matter of grave doubt at the present time.

In the spring of 1951, having achieved a state of development which permitted fairly regular creation and closure of interatrial septal defects in dogs, we were joined by Dr. Richard Varco in an effort to close such defects in failing hearts in children. The first case was reported by the group of us two years ago at the meeting of the American Surgical Association. It is of interest that that case concerned a patient with an atrial septal defect, and that the amount of return of blood to the right atrium from the intrinsic circulation of the heart was reported by us at that time to be in excess of 250 c.c. per minute also.

The second case was undertaken some weeks later. Under direct vision snug closure was easily accomplished. (Slide.) The difficulty in this case arose from several sources. This would apparently have been the first successful clinical closure of such a defect, if a member of the team had not failed to switch on a level control mechanism, an oversight which resulted in the forcing of oxygen into the aorta with obvious gas in the coronary and carotid arteries. The circulatory difficulties which resulted led to very severe pulmonary edema. However, continued support by virtue of femoral cannulas, to the extent of about 200 c.c. per minute, led to return of normal blood pressure, a strong heart beat, and an apparently normal electrocardiographic tracing. Although cerebral damage was apparent, the chest was closed, and the patient survived with the support of the oxygenator until the end of the day, at which time death occurred.

(Slide.) This experience and our finding of the disastrous effects of the inadequacy of chemical sterilization have led us to spend the intervening twenty months in design and construction of a new apparatus, which is virtually completely automatic; the blood-bearing unit of this apparatus can be autoclaved after complete assembly. We have been delayed many months by the move from Minneapolis to Brooklyn, but hope now for early resolution of some of the remaining problems.

I have visited with Dr. Clark and his associates, Dr. Gollan and Dr. Gupta of Yellow Springs, Ohio, and have been unable to escape the dread of the effect of gas bubbles in the blood returning to the septum. I should like to ask Dr. Helmsworth if he is satisfied that failure to find evidence of air embolism at autopsy is satisfactory removal of the possibility of this as a source of difficulty.

DR. JOHN H. GIBBON, Jr., Philadelphia.—I merely rise to say that while we have used this method of avoiding air embolism in the systemic arteries, we are not at all sure that it is going to prove eventually to be the best or the only method.

About six months ago Senning of Crafoord's laboratory published a paper in which he produced ventricular fibrillation deliberately in order to avoid having the left ventricle pump the air which it contained during the creation and closure of interatrial septal defects. Following the closure of the atrial septal defects he defibrillated the heart by electric shock. We were anxious to avoid, if possible, the use of such a potentially dangerous procedure in animals or patients, so we have used a fairly simple method.

This method consists of slipping a small plastic tube into the chamber of the left ventricle through a stab wound in the apex. The tube is connected to a source of mild suction. Contraction of the left ventricle then ejects any contained blood or air into this tube instead of into the aorta. The blood removed is returned to the extracorporeal circuit. Subsequently, after the operative procedure within the heart is completed, the tube can be quite simply withdrawn. We think it is important, when employing an open cardiotomy for the closure of septal defects, to have some method of being sure that air embolism will not occur in the systemic arteries.

DR. HELMSWORTH (Closing).—In answer to the question Dr. Dennis raised, of course, we cannot be certain. In the examination of dogs that have died after the experiment, very careful attention is given to the coronary vessels as well as the cerebral vessels; we feel that in the largest number of instances of oxygen embolization we can see collections of gas in those vessels. This finding is a very infrequent one at necropsy on animals in the most recent group of experiments.

VARIATIONS IN THE BRONCHOVASCULAR PATTERNS OF THE LEFT LOWER LOBE OF FIFTY LUNGS

MARTHA PITEL, M.S.,* AND EDWARD A. BOYDEN, PH.D.
MINNEAPOLIS, MINN.

THIS article represents the last of a series of studies analyzing the variations in gross structure of the bronchopulmonary segments of adult human lungs. Emphasis will be placed on the patterns assumed by the pulmonary arteries and veins of the left lower lobe. Yet because the patterns are largely determined by the arrangement of bronchi it has also been necessary to identify the bronchial variations in these fifty specimens. These, added to the ones described by Berg and Boyden in 1949, provide us with an incidence of variation based on 110 specimens.†

THE GENERAL VENOUS PATTERN

The left lower lobe is drained almost exclusively by the left inferior pulmonary vein. This is in marked contrast to the situation described by Boyden and Scannell in the right lung where the region of fusion of superior segment and upper lobe is partially drained by the posterior vein of the upper lobe in 54 per cent of specimens. A comparable arrangement (Fig. 2, *B*) was found in only 6 per cent of left lungs (Boyden and Hartmann, 1946). Presumably this is due to the fact that the left superior segment is rarely fused to the upper lobe, congenitally.

The prevailing pattern of the veins in the left lower lobe is illustrated in Plate 1. To expose the branches of the inferior pulmonary vein by dissection, a small vein is located in the hilum *(V^7b)* which runs beneath the pulmonary ligament (cf. Plate 2, left figure). This vein leads into the cleavage plane which separates the medial basal *(B^7)* from the posterior basal segment *(B^{10})*. After B^7 is reflected laterally, the big veins are encountered (right figure, Plate 2). These vessels fit snugly against the basal bronchi. In Plate 1 the inferior pulmonary has been retracted medially to show better the relationship of veins to bronchi.

In general, the venous pattern resembles that of the right lower lobe. The left pulmonary vein receives two main tributaries—V^6, which drains the superior segment (cf. Plate 3), and the common basal vein. The latter receives

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Abridgment of part of a thesis submitted by Martha Pitel to the faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Ph.D.

Aided by grants from the Medical Research Funds of the Graduate School as part of a long-term program under the direction of E. A. Boyden.

Received for publication July 10, 1953.

*Research Fellow, United States Public Health Service.

†The authors are indebted to Dr. Ronald M. Ferry, Jr., and Dr. Dennis Kane for a preliminary dissection of ten of the fifty specimens showing the bronchovascular patterns.

two main tributaries—the superior basal (*S*) and the inferior basal (*I*).* These, in turn, receive the segmental and intersegmental veins.† However, there is one striking difference between the veins of the two lower lobes. On the right, the superior basal vein is more commonly formed by V^8 and V^9, and the inferior basal by V^{10}. On the left, the reverse is true: V^9 and V^{10} more commonly unite to form the inferior basal (Table I). Also, the common trunk of V^9 and V^{10} is shorter (Plate 1), so that occasionally V^9—or a subsuperior vein which is usually tributary to it, *VX* (10)*—forms the middle member of a trifurcation (Table I). Then we may recognize a common basal vein which is formed by three major trunks—a superior basal, a posterior basal, and an inferior basal vein (cf. Plate 4, #12). Such an arrangement has not been observed in the right lower lobe.

In constructing Table I, small tributaries of the basal veins such as V^7, or the usually small *VX* (9)* and *VX* (10)*, etc., have been omitted. Yet, it has been necessary to recognize "split" types, as in the right lower lobe. Thus, in 18 per cent of our 50 specimens, V^{10}, V^9, or V^8 is represented by two veins, one of which joins the superior basal vein, the other of which joins the inferior basal vein. In the right lower lobe this occurred in 62 per cent of 50 specimens.

TABLE I. PATTERN OF BASAL VEINS

	SUP. BASAL	POST. BASAL	INF. BASAL	PERCENTAGE	
Bifurcations					94%
Uncomplicated types	V^8		V^{9+10}	44%	
	V^{8+9}		V^{10}	32%	
"Split" types	V^{8+9}		V^{9+10}	4%	
	$V^{8,9,10}$		V^{10}	8%	
	V^8		$V^{8,9,10}$	6%	
Trifurcations					6%
	V^8	V^9	V^{10}	2%	
	V^{8+9}	VX^*	V^{10}	4%	

Last, displaced lingular veins occurred in only five specimens (10 per cent). Four of these emptied into the inferior pulmonary vein (two being associated with V^7), and one into the superior basal. (Boyden and Hartmann found the same percentage of displaced lingular veins.) In general, it is clear that the left inferior pulmonary vein is much less variable in its mode of formation than the right inferior pulmonary. (See Table III of Ferry and Boyden.)

Of special interest is the specimen illustrated in Fig. 3. This is the only case in which the basal veins appeared to cross the anterior surface of the lobe superficially. This is due to the fact that B^7 arises as a displaced ramus *(BX^7)* of the lateral basal bronchus *(B^9)*.

THE SUPERIOR SEGMENT *(B^6)*

1. *New Observations on the Bronchial Pattern.*—Before discussing the blood supply of the segment we shall briefly review the bronchial pattern of the segment and then present new observations on its variability.

*For discussion of terminology, see Ferry and Boyden, 1951.

†According to the system devised by Boyden in 1945, the vein which lies on the clockwise side of a given left bronchus (as viewed from in front) receives the same number as that bronchus (cf. Plate 1).

TABLE II. SUMMARY OF SUPERIOR SEGMENTAL BRONCHUS (B^6)

	PITEL AND BOYDEN (50 SPEC.)	BERG AND BOYDEN (60 SPEC.)	TOTAL (110 SPEC.)	
I. B^6 arises as a single trunk				
1. B^6 bifurcates into				83.7%
1) B^6a and $B^6b + c$	42%	43%	42.7%	
2) B^6b and $B^6a + c$	32%	27%	29.1%	
3) B^6c and $B^6a + b$	2%	10%	6.4%	
4) Bizarre patterns (split B^6b)	6%	5%	5.5%	
2. B^6 trifurcates into				15.4%
1) B^6a; B^6b; B^6c	14%	15%	14.5%	
2) B^6a1; $B^6a2 + c$; B^6b	2%	0%	0.9%	
II. B^6 arises as two separate trunks				0.9%
$B^6b + c$ and B^6a (Plate 4, #7)	2%	0%		

The superior or "apical" bronchus of the left lower lobe has three main branches: a medial ramus *(B^6a)*, a superior ramus *(B^6b)*, and a lateral ramus *(B^6c)*. The present study confirms the observation of Berg and Boyden that the most common pattern (43 per cent) is a bifurcation having the formula "B^6a and $B^6b + c$" (cf. Table II and Plate 5). By virtue of the fact that B^6a extends downward along the paravertebral surface toward the diaphragm (Table III), the left superior segment caps the lower lobe obliquely, whereas on the right side it is prevailingly horizontal. This distinction is best shown in colored plates of injected specimens (1949 articles) and explains why resection of the superior segment can be more difficult on the left side.

TABLE III. LOWEST EXTENT OF MEDIAL RAMUS (B^6a) ON THE PARAVERTEBRAL SURFACE

	PITEL AND BOYDEN (50 SPEC.)	BERG AND BOYDEN (60 SPEC.)	TOTAL (110 SPEC.)
Extends two-thirds or more of distance to diaphragm	38%	57%	48.2%
Half-way to diaphragm	46%	37%	40.9%
One-third of distance to diaphragm	10%	3%	6.4%
Fails to reach paravertebral surface*	6%	3%	4.5%

*In 12% of the 110 specimens the upper portion of the paravertebral surface is supplied by an accessory branch of B^6b, which is then designated BX^6a1. In 4.5% the accessory branch replaces B^6a1; in the remaining 7.5% it supplements B^6a1.

In the present series of specimens we have encountered a new complication—namely the displacement of B^6a to a position on the basal bronchus 14 mm. below the main orifice of B^6 (see Specimen #7, Fig. 1). That this displaced ramus is not a medial subsuperior bronchus *(B^*)*—as one might first surmise—is made clear by a comparison of Specimens #7 and #12 in Plate 4. In #12, B^6a (with its superficial and deep branches, B^6a1 and B^6a2) is obviously the medial ramus of a bifurcating B^6. In #7, the bronchus with branches labeled B^6a2 and B^6a1 distributes to virtually the same territory. Therefore, it represents a displaced B^6a. Also, as in #12, its artery *(AX^6a)* arises atypically from A^{10}, but at the same level as the displaced artery to the lateral ramus, namely AX^6c.

This finding of two orifices for the left superior segment is unique in our experience. On the right side such an arrangement has been noted in 6 per

cent of 100 specimens (Table II of Ferry and Boyden). On that side, however, the lower orifice represents ramus B^6c (see Fig. 1 of these authors).

In the present study of the left lobe we have also encountered, for the first time, two instances of a subsuperior bronchus *(B*)* which supplies the paravertebral surface. Previously Neil and his co-workers had mentioned that such a medial stem existed on the left side. An example of this is now shown in Plates 2 and 3. In the left-hand figure of Plate 2, B^* occupies a paravertebral position. Why, then, should it not be interpreted as a displaced B^6a? The first reason is that a B^6a2 is already present in its usual place. Second, only a portion of B^* supplies the superficial paravertebral surface. Most of it extends posteriorly (see truncated lobe on right side of Plate 2). It is interesting to note, however, that since its orifice is only 12 mm. below the orifice of B^6, the bronchoscopist would not be able to distinguish between a medial subsuperior and an inferomedially displaced second orifice of B^6.

The question will also be raised as to why a high, *laterally* directed subsuperior, which frequently supplies the anterior surface below a high B^6c, should not be interpreted as an inferiorly displaced B^6c, especially since this is the form of duplication that occurs in the right superior segment. In answering this, attention is called to the deceptive specimen presented in Plate 5. Twelve millimeters below the keel of B^6, there arises a B^* with deep branches passing to the posterolateral surface and superficial branches supplying the anterior surface. In the absence of B^6a, the superficial ramus is interpreted as a displaced B^6a *(*BX^6a from B^**)*. But inferior to these superficial branches is a vein (here labeled VX^6a) which empties into the superior vein *(*V^6*)*. Since the bronchus in question is cut off by a branch of the superior vein, why should this bronchus not be identified as a displaced B^6c? The answer is that it is too low, also that this is the direction which the subsuperior bronchus usually assumes on the left side (see footnote, Table VII). Therefore it is a subsuperior bronchus carrying certain anterior basal branches.

With this introduction, we may now proceed to consideration of the arrangement of blood vessels in the superior segment.

2. *The Arteries of the Superior Segment (*A^6*).*—In a previous analysis of the left upper lobe it has been noted that the left pulmonary artery crosses the bronchus of the left upper lobe to enter the floor of the interlobar fissure. As it makes the turn it gives off a varying number of superior and posterior arteries to the superior division segments of the upper lobe (Fig. 2). Somewhat below these, on its medial side, appears a cluster of arteries to the lingular segments ($A^{4,5}$, Fig. 2, *B*). Unexpectedly, the first artery to the lower lobe arises between these two groups (A^6, Fig. 2), that is, above some of the arteries of the upper lobe. For this reason, Kent and Blades early pointed out the necessity of ligating the arteries individually in lobectomies of either the left upper or lower lobes. Even when there are two separate arteries to the superior segment ($A^6a + b$ and A^6c, Fig. 2, *C*) this relationship still holds, as a rule. In this respect, therefore, the left pulmonary artery differs in its mode of branching from the artery of the right lung. For on that side, when there are two or more arteries to the superior segment, the lower one usually arises below the middle

lobe artery—or the second middle artery, if there are two. (See Fig. 3 of Boyden and Hamre.)

As indicated in Table IV, the left superior segment was found to be supplied by two arteries in about one-third of our fifty specimens. The incidence of this variation is thus twice as great as in the right superior segment (16 per cent, Ferry and Boyden). Furthermore, the complex types of branching are somewhat more numerous on the left—28 per cent (Table IV) as against 24 per cent on the right.

TABLE IV. ARTERIAL SUPPLY OF SUPERIOR SEGMENT

I. Segment supplied by *single* artery bifurcating into			64%
1. Simple types		48%	
1) A^6a and $A^6b + c$	22%		
2) A^6b and $A^6a + c$	24%		
3) A^6c and $A^6a + b$	2%		
2. Complex types (with displaced branches)		16%	
II. Segment supplied by *two* separate arteries			34%
1. Simple types		22%	
1) A^6a and $A^6b + c$	12%		
2) A^6b and $A^6a + c$	4%		
3) A^6c and $A^6a + b$	6%		
2. Complex types (with displaced branches)*		12%	
III. Segment supplied by *three* separate arteries (One of the three, AX^6a, came from A^{10})			2%

*In 5 of the 6 specimens, A^6a1 (or A^6a2) arose atypically from A^{10}.

Of special interest are the 12 per cent of specimens in which A^6a, or one of its two branches, arises from a displaced position on A^{10}, the artery to the posterior basal segment (cf. AX^6a2, Plate 5; AX^6a, Plate 4). Such an arrangement was never found in the right lower lobe. Also, in 14 per cent, A^6a1 arose as a displaced branch of A^6b (cf. AX^6a1, Plate 2). These aberrations are presumably correlated with the greater paravertebral extent of B^6a in the left lung. In view of these variations, we were surprised to find that the arterial pattern corresponds to the bronchial pattern more frequently in the left superior segment than in the right. For in only 36 per cent do the arteries cross the subsegmental planes, whereas on the right side the arteries fail to reproduce the bronchial pattern in 44 per cent of specimens. Nevertheless, it would seem that resection of subsegmental portions of the superior segments would be somewhat hazardous. This is accentuated by the variability of the veins.

3. *The Veins of the Superior Segment (V^6).*—With few exceptions the veins of the superior segment drain into a large trunk (V^6) which lies on the median side of the left upper lobe bronchus (Plate 1). As it swings around from behind the upper lobe bronchus, it varies in the depth of its position. Prevailingly (56 per cent), it emerges from the deep side of B^6a (Plate 5). In another 30 per cent, V^6 or its main branch passes superficially to B^6a (Specimen #12, Plate 4); and in the remaining 14 per cent of specimens it courses between the two branches of B^6a (Plate 2).

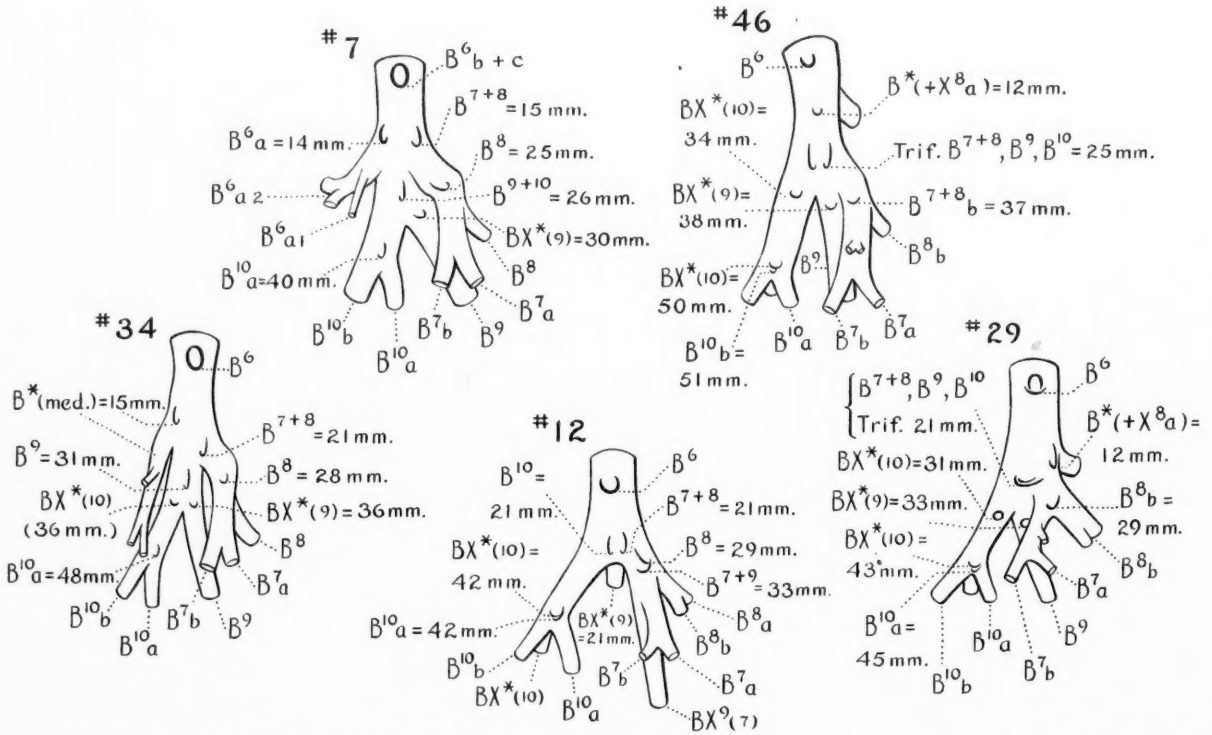

Fig. 1.—Bronchial trees of specimens of left lower lobe selected to illustrate certain variations. The trees are drawn as if transparent in order to show position of keels. Distances are measured from the orifice (keel) of B^6. *Specimen* #7 (cf. Plate 4), illustrating only case of double origin of the left superior bronchus (B^6). *Specimen* #46 (cf. Plate 5), illustrating common form of trifurcation. *Specimen* #34, illustrating one of two specimens having a medial (paravertebral) subsuperior bronchus (B^*). *Specimen* #12 (cf. Plate 4), illustrating rare type of trifurcation into B^{10}, BX^* (9), and B^{7-9}. *Specimen* #29, illustrating laterally placed subsuperior bronchus (B^*) invading the anterior basal segment (cf. Plate 5).

Usually V^6 has three principal tributaries. Of these, the middle one *(V^6b)* is somewhat vertically oriented and emerges from the depths of the segment. This relationship is well shown in Plate 3 (see large unlabeled branch of V^6 that lies parallel and just inferior to B^6b). The other two, V^6a and V^6c, are usually intersegmental veins which separate the superior segment from adjacent zones. Commonly these are horizontally placed. In Plate 3, for instance, V^6a2 separates the superior segment from the subsuperior zone (see bronchus associated with A^*). In Plate 2 the same vein (of the same specimen) is seen on the bottom of the upturned superior segment. On the lateral side, the vein which separates the superior from the anterior basal segment, or its displaced lateral branch *(BX^6a)*, is V^6c. This is well shown in Specimen #7 (Plate 4) and in Plate 5. However, these intersegmental veins are not always present. In 10 per cent there is no large V^6a branch but only scattered venous twigs, and in another 10 per cent V^6c is wholly or virtually absent (Plate 2).

The mode of branching is also variable. In fact the vein often exhibits a shrublike appearance, so that in analyzing its pattern one has to deal with just the major tributaries. The prevailing patterns have the formula V^6b and $V^6a + c$ (36 per cent) or V^6a and $V^6b + c$ (32 per cent). In another 12 per cent there was a "splitting" of V^6a or V^6b—that is, one or the other of these rami were represented by two large branches widely separated from each other. In another 20 per cent (as noted earlier) either V^6a or V^6c were defective. In the remaining 2 per cent the vein was formed by the two tributaries V^6c and $V^6a + b$.

Because of these variations the mode of branching of V^6 was observed to follow the bronchial pattern in only 22 per cent of specimens. Finally, in a comparison of the venous drainage of the left and right superior segments, the principal difference would be found in the greater development of V^6a in the left lung. This may be correlated with the greater prominence of B^6a on the left side. It will be recalled that the major ramus of the right segment is B^6c, and that in 60 per cent of right superior segments V^6 was formed by only two major tributaries—V^6b and V^6c.

THE BASAL SEGMENTS

1. *General Mode of Division of Basal Bronchi and Arteries.*—After the lower lobe bronchus gives off the dorsal branch that supplies the superior segment *(B^6)*, it continues as an undivided trunk for an average distance of 17 mm. We have named this portion of the bronchial tree the "basal trunk" (Fig. 3).* In nearly four-fifths of the lobes, this trunk divides into two stems—commonly B^{7+8} and B^{9+10} (62 per cent of 110 specimens). In the remaining fifth it branches into three stems (Table V).

The corresponding portion of the left pulmonary artery—the "basal artery"—has the following patterns: in 42 per cent it divides into A^{7+8} and A^{9+10} (cf. Fig. 2, *A*), and in 4 per cent it trifurcates (cf. Plate 4, #7)†; but

*Its range in length is from 8 to 27 mm. with peaks at 15 and 20 mm. In defining this as an undivided stem we have disregarded the occasional presence of a high subsuperior offshoot *(B^*)* which occurs in 14 per cent of 50 specimens (cf. Plate 2, middle figure).

†In this classification the numerous and variable arteries to the subsuperior segments *(A^*)* have been disregarded (cf. Fig. 2, *B*). These will be discussed later.

TABLE V. MODE OF BRANCHING OF BASAL TRUNK†

	PITEL AND BOYDEN (50 SPEC.)	BERG AND BOYDEN (60 SPEC.)	TOTAL (110 SPEC.)	
I. Basal trunk bifurcates				77.3%
1. Into B^{7+8} and B^{9+10}†	56%	66.7%	61.8%	
2. Into bizarre patterns	10%	20.0%	15.5%	
1) $B^{7+8}a$ and $B^{8}b + B^{10}$ (B^{9} absent)	0%	1.7%	0.9%	
2) B^{7} and rest	4%	10.0%	7.3%	
3) B^{8} and rest	0%	1.6%	0.9%	
4) B^{10} and rest	6%	6.7%	6.4%	
II. Basal trunk trifurcates				22.7%
1. Into B^{7+8}; B^{9}; B^{10}	30%	13.3%	20.9%	
2. Into $B^{8} + B^{7+9}$; BX^{*} (9); B^{10} or B^{7+8}; BX^{*} (9); B^{10+9}	4%	0.0%	1.8%	

†For details of variation within a given stem, such as those for B^{7+8}, B^{9+10}, and so forth, see following tables.

in the remaining 54 per cent it divides very unevenly—into A^{10} and the rest (46 per cent, cf. Fig. 2, *C*), A^{8} and the rest (6 per cent, cf. Fig. 2, *B*), or $A^{10}b$ and the rest (2 per cent).

The peculiar grouping together of arteries 7, 8, and 9 (46 per cent) may be explained largely by peculiarities of the bronchial pattern in these specimens—namely by the displacement of B^{9} to a position on B^{7} (8 per cent, cf. BX^{9}, Fig. 1, #12 and Plate 4, *B*), by the trifurcation of the basal bronchus (28 per cent, cf. #46, Fig. 1), or by the shortness (3 to 7 mm.) of the common

TABLE VI. THE MEDIAL BASAL BRONCHUS (B^{7})

	PITEL AND BOYDEN (50 SPEC.)		BERG AND BOYDEN (60 SPEC.)		TOTAL (110 SPEC.)	
I. Origin of B^{7}						
1. Arises in common with B^{8} (or ramus)		96%		86.6%		90.9%
1) B^{7} lies anterior to B^{8}	66%		47%		55.5%	
2) B^{7} lies in same plane as B^{8}	30%		35%		32.7%	
3) B^{7} lies deep to B^{8}	0%		5%		2.7%	
2. Arises separately from basal trunk		4%		10%		7.3%
3. Absent as such (displaced to B^{9})		0%		3%		1.8%
II. Distribution of B^{7}						
1. Supplies whole width* of anterior surface	28%		61.7%		46.4%	
2. Supplies medial half or third of anterior surface	36%		21.7%		28.2%	
3. Supplies anterior and sector of costal surface	36%		10.0%		21.8%	
4. Supplies one-half of anterior surface (one ramus absent)	0%		3.3%		1.8%	
5. Absent as such	0%		3.3%		1.8%	
III. Occurrence of large diaphragmatic ramus ($B^{7}c$)						
1. Arises as branch of $B^{7}b$	26%					
2. Arises as branch of $B^{7}a$	16%					
3. Arises from common trunk of B^{7}	16%					
4. Arises from B^{9} (as $BX^{7}c$)	2%					
5. Represented by small branches	40%					

*As measured along inferior margin from pulmonary ligament to lateral margin of lobe.

trunk of B^9 and B^{10} (8 per cent, cf. Plate 5). This accounts for the pattern in eighteen of the twenty-two specimens in which A^7, A^8, and A^9 have a common stem. Nevertheless, the fact remains that in two-thirds of all specimens the basal artery has a different mode of branching than the associated basal bronchus.

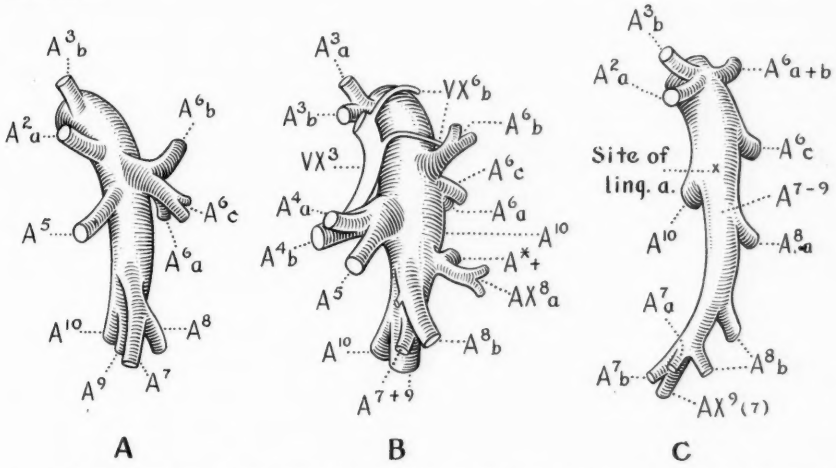

Fig. 2.—Sketches of the interlobar portion of the left pulmonary artery to illustrate relation of arteries of the superior segment (A^6) to the lingular arteries of the upper lobe (A^4 and A^5). *Specimen A*, illustrating single A^6 at level between artery to the anterior segment (A^4a) and A^5. The basal artery divides conventionally into A^{7+8} and A^{9+10}. *Specimen B*, illustrating two superior segmental arteries lying above level of A^4 and A^5, also atypical division of basal artery into A^8b and $A^{7+9} + A^{10}$. *Specimen C*, illustrating absence of lingular arteries on interlobar portion of pulmonary artery and the prevailing pattern of division of basal artery into A^{10} and A^{7-9}.

2. *The Medial Basal Segment (B^7).*—

The bronchial pattern: As summarized in Table VI, the medial basal bronchus has the following characteristics. It arises in common with B^8 (or one of its rami) in approximately 91 per cent of 110 specimens (cf. Plate 1).* In 7 per cent it arises independently from the medial portion of the basal trunk, as does its homologue of the right lower lobe (see Fig. 5 of Berg and Boyden). In 2 per cent it is absent as such (see Fig. 3). It distributes prevailingly (in 46 per cent) to the whole width of the anterior surface of the base of the lobe (Plate 5), or (in 30 per cent) it may supply less than that (Plate 4, #7), or (in 22 per cent) it may even extend onto the costal surface (Plate 4, *C*).

The diaphragmatic surface of the segment is supplied prevailingly (in 40 per cent) by small branches of B^7a and B^7b, or (in 42 per cent) by a large branch of either B^7a or B^7b, or (in 16 per cent) by a single large stem (B^6c) which arises from the main trunk of B^7, before the latter divides into the usual medial and lateral rami (cf. Fig. 3). A discussion of the reasons why the medial basal bronchus should be considered to have the status of at least a subsegmental bronchus (and perhaps a segmental one) may be found in the article by Boyden, 1953.

*The average length of this common trunk in 28 specimens is 8.5 millimeters.

The arterial pattern: Since B^7 arises in common with B^8 or one of its branches, in 91 per cent of specimens, it would be expected that A^7 would usually have a common origin with A^8 or one of its branches, and such is the case in all but 8 per cent of fifty specimens (cf. Plate 4, #12). Similarly, since B^7 at its point of origin lies in a plane superficial to B^8 (66 per cent of fifty specimens) one would assume that A^7 would cross in front of B^8 in the majority

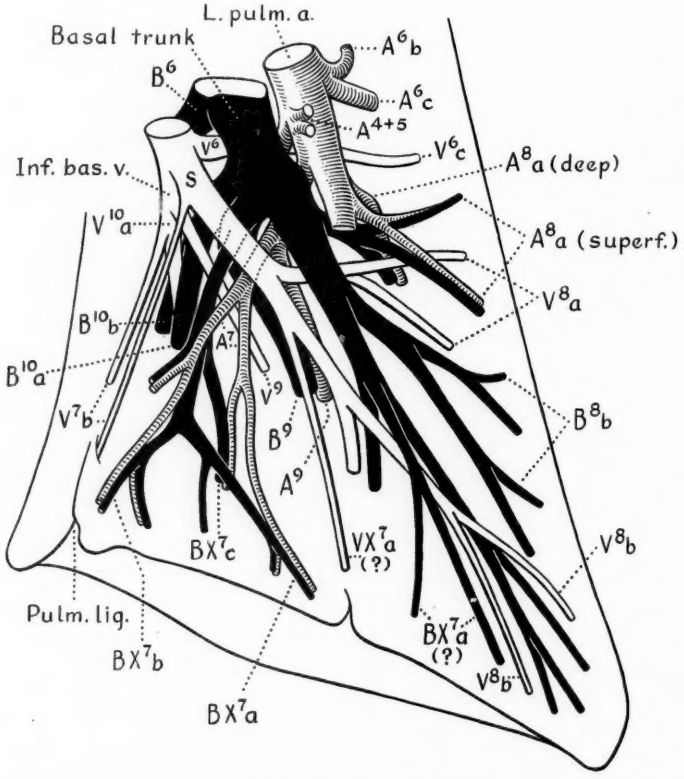

Fig. 3.—Sketch of dissected anterior surface of left lower lobe of a unique specimen. (Dissection by S. H. Robinow.) Superior basal vein passes anterior to all bronchi except B^8b. B^7 (the medial basal) is absent as such and originates as a displaced bronchus (BX^7) from the proximal portion of the lateral basal bronchus (B^9). The basal trunk trifurcates into B^8, B^{7+9}, and B^{10}. Note high position of A^6 above level of lingular arteries (A^{4+5}). The A^6c branch is hidden behind A^6b. The territory supplied by BX^7 is narrower than usual, and some of the branches of B^8b have invaded its territory (BX^7a?).

of specimens, and such is the case (cf. Plate 4, #7). However, in 26 per cent, A^7 passes deep to the anterior basal bronchus. Such specimens are dangerous from the standpoint of resection of segments, for in about one-half of these specimens the artery to the lateral basal segment (A^9) arises anomalously from the deep side of A^7. In such lobes the ligation of a supposedly single artery could well occlude two segmental arteries instead of the anticipated one. An example of a displaced artery (AX^9), arising from the deep side of A^7b, is shown in Fig. 2, *C* (AX^9 from ^{7}).

The venous pattern: The veins of the medial basal segment are small vessels ranging in number from one to four, and occasionally there are more. Typically, there are two (36 per cent of specimens). The first of these *(V^7a)* runs between the two rami of the segment (Plate 5) and empties, in two-thirds of the specimens, into V^8b—the intersegmental vein which separates the medial basal from the lateral basal segment (Plate 5). However, it may drain into the common basal, the superior basal, or even the inferior basal or one of its branches (V^9, Plate 2, right figure).

The second vein, V^7b (Plate 4, #7), is the intersegmental vein which separates the medial basal from the posterior basal segment *(B^{10})*. This is the vein which usually lies deep to the pulmonary ligament (Plate 2, left figure) and leads into the cleavage plane which separates the shallow medial basal segment from the underlying segments, B^{10} and B^9. Typically, V^7b drains into the common basal vein (Plate 4, #7), but almost as frequently into the inferior basal (Plate 2, right figure). Atypically, it discharges into the superior basal vein or one of its branches.

When there are more than two veins draining the segment (44 per cent) there may be two V^7*a*s and one V^7b, or vice versa, or two of each. The remaining 20 per cent of specimens are characterized by the presence of a single V^7 having *a* and *b* rami. This may be the only vein, or it may be supplemented by a second vein, V^7a or V^7b. On its deep side the medial basal segment is usually separated from the lateral basal segment by tributaries of V^9 (Plate 2, right figure). Thus in resecting the medial basal segment lateralward, one could enter the pulmonary ligament to V^7b, thence along the tributaries of V^9, and thence to V^8b, the vein which separates the medial basal from the anterior basal segment.

3. *The Anterior Basal Segment (B^8).—*

The bronchial pattern: In contrast to the median basal bronchus with which it is so frequently associated, the anterior basal bronchus is a highly variable and frequently defective stem, both in its mode of branching and in its distribution. As summarized in Table VII, one or the other of its two rami was missing in 17 per cent of 110 specimens. The territory normally supplied by its lateral ramus (B^8a, Plate 2) is either invaded by neighboring bronchi (cf. Plate 4, #7) or wholly supplied by others (Plate 5) in 35 per cent of specimens. Its basal ramus, which normally reaches the diaphragmatic surface of the lobe (B^8b, Plate 4, #7), fails to reach the base of the lung in 19 per cent of specimens (cf. Plate 4, *C*). In the present study we have become aware of the frequency of another variation in the mode of branching of the anteromedial basal trunk—namely, the high origin of B^8a on the common stem of B^{7+8}. As a result, the trunk divides unevenly into B^8a and $B^{7+8}b$. This was observed in 24 per cent of fifty specimens (Plate 2, left figure). It is not surprising, therefore, that the arteries supplying the segment should have a complicated pattern.

The arterial pattern: The artery to the anterior basal segment arises separately from the basal artery in only three cases (6 per cent of fifty specimens). In two of these A^8 arises high up on the basal artery. Lower down the latter then divides into A^7 and A^{9+10}, or A^{7-9}, $A^{10}a$, and $A^{10}b$. In the third case

TABLE VII. THE ANTERIOR BASAL BRONCHUS (B^8)

	PITEL AND BOYDEN (50 SPEC.)		BERG AND BOYDEN (60 SPEC.)		TOTAL (110 SPEC.)	
I. Origin of B^8						
1. Arises wholly or in part with B^7		96%		86.6%		90.9%
1) B^8 has typical pattern (Plate 1)	56%		65.0%		61.0%	
2) B^8a arises higher, $B^{7+8}b$ lower, on stem	18%*		8.3%		12.7%	
3) B^8a absent (stem consists of $B^{7+8}b$)	16%†		8.3%		11.8%	
B^8a replaced by ramus of BX^* *(9)*,	8%					
B^8a replaced by ramus of B^*,	6%					
B^8a replaced by ramus of B^9,	2%					
4) B^8 carries displaced B^9 (In 2%, B^8a arises high on stem) (In 2%, B^8a replaced by B^*)	6%		1.7%		3.6%	
5) B^8b absent (stem consists of $B^{7+8}a$) (B^8b replaced by ramus of B^9)	0%		3.3%		1.8%	
2. B^8 arises alone from basal trunk		4%		13.4%		9.1%
II. Distribution of B^8a and B^8b						
1. B^8a alone supplies upper half of segment						64.6%
2. B^8a assisted or replaced‡						35.4%
1) By BX^* *(9)*	22%		13.3%		17.3%	
2) By B^*	18%		13.3%		15.4%	
3) By B^9	2%		0.0%		0.9%	
4) By BX^* *(10)*	4%		0.0%		1.8%	
	46%		26.6%		35.4%	
3. B^8b reaches basal surface of lobe		78%		83.3%		80.9%
4. B^8b fails to reach basal surface		22%		16.7%		19.1%

*Total is 20% of 50 specimens (add line 16 of table, and cf. Plate 2).

†Total is 18% of 50 specimens (add line 18 of table, and cf. Plate 5).

‡B^8a assisted by other bronchi in 21.8% (cf. Plate 4), replaced by others in 13.6% of 110 specimens (cf. Plate 5).

A^8a was absent as such, and A^8b arose high up on the basal artery. Lower down the latter divided into A^{10} and $A^7 + A^9 + AX^*$ *(9)*. In the remaining 94 per cent, A^8 or its rami were branches of the anteromedial basal artery. Accordingly the patterns of A^8 will be discussed in relation to the parent artery, A^{7+8} (cf. Plate 4, #7).

The three major patterns are shown in Fig. 4. Diagram *A* represents the prevailing type (20 per cent) in which the parent artery divides into A^7 and A^8.* The arrangement conforms to the bronchial pattern of the corresponding specimens in only 16 per cent of cases, yet 42 per cent of the bronchial trees have this conventional pattern.

In the second type (*B*, Fig. 4), A^8b arises separately as a branch of A^7 in 22 per cent of specimens. Rarely this type may have two A^8as (Plate 2, left

*In another 10 per cent of these specimens, A^{10} arose higher up than is shown in the diagram and A^9 arose coordinately with A^{7+8}.

figure). In only seven of these eleven specimens is the arterial pattern duplicated by the corresponding bronchi.

In the third type (*C*, Fig. 4) A^8a is absent as such in all five, and A^9 now arises coordinately with A^{7+8}. In each case the bronchial pattern is different.

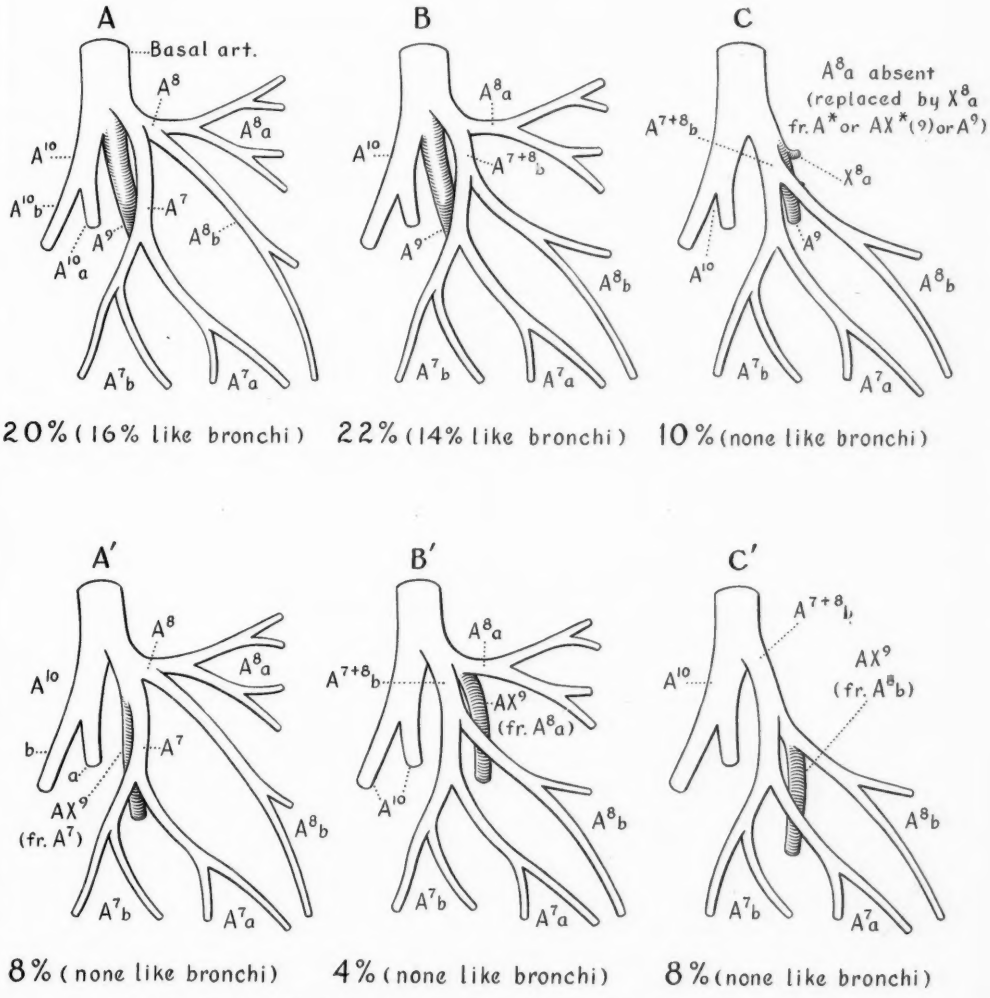

Fig. 4.—Diagram illustrating variations in pattern of the anteromedial basal artery (A^{7+8}). Diagram *A* shows the conventional pattern of arteries of the left lower lobe. In another 10 per cent A^{7+8} presents the same pattern but the artery arises collaterally with A^9 (A^{10} coming off by itself). In diagram *B*, A^8a arises separately from the common stem. In *C*, A^8a is absent as such, and it originates as a displaced branch of A^9 which now arises collaterally with A^{7+8}. In the lower row of figures, A^{7+8} has the same pattern as in the figure above it, but this time A^9 is displaced and originates from the deep side of either A^7 (Fig. *A'*), A^8a (Fig. *B'*), or A^8b (Fig. *C'*). In an additional specimen, A^9 arose from the main stem of A^8, making a total of 22 per cent in which A^9 is displaced.

In the lower row of diagrams (*A'*, *B'*, *C'*, Fig. 4) similar arterial patterns are again shown, this time representing specimens complicated by displacement of the lateral basal artery (*A^9*) to a position on the deep side of *A^7*, *A^8a*, or *A^8b*. In none of these does the arterial pattern conform to the bronchial plan. In addition to these ten there is another specimen in which *A^9* is displaced to the main stem of *A^8*. This makes a total of 22 per cent in which *A^9* arises anomalously from the deep side of *A^7*, *A^8*, *A^8a*, or *A^8b*. Resection of the anteromedial basal segment in these specimens, therefore, could have jeopardized the blood supply of the lateral basal segment.

Finally it may be stated that only one-third of the anteromedial basal arteries (32 per cent) conform to the respective bronchial patterns.

TABLE VIII. THE LATERAL BASAL BRONCHUS (*B^9*)

	PITEL AND BOYDEN (50 SPEC.)		BERG AND BOYDEN (60 SPEC.)		TOTAL (110 SPEC.)	
I. *B^9* arises in conjunction with *B^{10}*		88%		90%		89.1%
1. From a trunk dividing into *B^9* and *B^{10}*	54%*		66.7%		60.9%	
2. Collaterally with *B^{10}* and *B^{7+8}*	30%		13.3%		20.9%	
3. From a common trunk, *B^{8-10}*	4%		10 %		7.3%	
II. *B^9* absent as such (displaced)		12%		10%		10.9%
1. Replaced by ramus of *B^7*	2%		6.7%		4.6%	
2. Replaced by ramus of *B^{7+8}*	2%		0.0%		0.9%	
3. Replaced by ramus of *B^8*	2%		0.0%		0.9%	
4. Replaced by ramus of *B^8b*	2%		0.0%		0.9%	
5. Replaced by ramus of *$B^{10}a$*	4%		1.7%		2.7%	
6. Replaced by rami of *B^7*, *B^8*, *B^{10}*	0%		1.6%		0.9%	
III. *B^9* gives off high lateral branch, *BX* (9)*		74%†		66.7%		70%

*To make this percentage conform to the 56% in Table V (Item I, 1), 2% must be taken from Item II, 5 of this table. The average length of this common trunk is 9 mm.

†In an additional 4%, *BX* (9)* is the middle limb of a trifurcation (cf. Fig. 1, #12, and Plate 4). In another 2% it is displaced onto *B^8a*.

4. *The Lateral Basal Segment (*B^9*).—*

The bronchial pattern: With the exception of *B^8*, the lateral basal bronchus is the most variable of the basal bronchi, being absent as such in approximately 11 per cent of 110 specimens. Prevailingly, as summarized in Table VIII, it arises exclusively with *B^{10}* from a common stem *B^{9+10}*. This arrangement holds in three-fifths (60.9 per cent) of 110 specimens (cf. Plate 4, #7). In an additional three-tenths (28.2 per cent), it arises in conjunction with *B^{10}* but also with other bronchi. Soon after it is formed it gives off a high posterior lateral ramus, *BX* (9)*, which contributes to the subsuperior zone (Table IX). This occurs in 70 per cent of 110 specimens. Its keel arises from 3 to 17 mm. below the keel of *B^9*, at an average distance of 9.3 mm.

The arterial pattern: Since the lateral basal bronchus originates from a common trunk with *B^{10}* in three-fifths of fifty specimens, it would be expected that the arteries would have the same conventional arrangement, but such a pattern is realized in only 42 per cent (Fig. 2, *A*). The striking feature is the separate origin of *A^{10}* in 46 per cent of specimens, Fig. 4, *B* (and the occurrence of other variations in an additional 12 per cent). Accordingly *A^9*

TABLE IX. DISTRIBUTION OF BX* *(9)*

	PITEL AND BOYDEN (50 SPEC.)	BERG AND BOYDEN (60 SPEC.)	TOTAL (110 SPEC.)
I. Sector			
1. Posterolateral only	34%	40%	37.3%
2. Posterolateral and lateral	42%	14%	26.4%
3. Posterior and posterolateral	2%	8%	5.4%
4. Posterior, posterolateral, and lateral	2%	5%	3.6%
	80%	67%	72.7%
II. Angle made with anterior surface of lobe			
1. 40-60 degrees (posterolateral)	42%	45%	43.7%
2. 20-35 degrees (lateral)	36%	12%	22.7%
3. 70-90 degrees (posterior)	2%	10%	6.3%
	80%	67%	72.7%

must originate prevailingly with A^7 and A^8, either as collateral branches of these and their rami (36 per cent) or as displaced branches of these (22 per cent, cf. Fig. 4, *A'*, *B'*, *C'*). The clinical importance of the latter, from the standpoint of resection of the anteromedial basal segment, has already been pointed out.

The venous pattern: The intersegmental vein *(V^9)*, which lies on the clockwise side of B^9, separates the lateral basal from the posterior basal segment (Plates 1 and 2). As summarized in Table I, V^9 empties either into the inferior basal vein (50 per cent of specimens), or into the superior basal (44 per cent), or into both (4 per cent). In one instance, it was the middle member of a trifurcating inferior pulmonary vein.

5. *The Posterior Basal Segment (B^{10}).*—

The bronchial pattern: In contrast to the other basal bronchi of the left lower lobe, B^{10} is the most constant both in its mode of division and in its distribution. It arises alone from the basal trunk, that is, as one of the two stems of the principal bifurcation, in 6 per cent of 110 specimens (Table X). In one-fifth of the lobes (20.9 per cent), it is one of the members of a trifurcation (Plate 5); in three-fifths—the prevailing pattern—it originates with B^9 from the common trunk B^{9+10} (Plate 2, middle figure).

Before the posterior basal divides into its terminal rami ($B^{10}a$ and $B^{10}b$—the laterobasal and mediobasal rami) it gives off from one to three *dorsal branches, BX* (10)*. These are present in about 85 per cent of the 110 specimens. The highest of these (or, prevailingly, a single dorsal ramus) supplies the posterior portion of the subsuperior zone (Table XI and Plate 4, *C*). Its keel arises from 3 to 21 mm. below the keel of B^{10}, at an average distance of 8.7 mm. It thus originates at about the same level as *BX* (9)*—the other most constant subsuperior bronchus.

The second type of collateral branch from the stem of B^{10} is the *paravertebral ramus* (cf. Berg and Boyden, Plate 4). This occurs in a third of 110 specimens (32.8 per cent). This may arise in conjunction with the first or second dorsal ramus, *BX* (10)*, or separately (12 per cent of 50 specimens). The average distance from the keel of B^{10} is 18 mm.—well above the point where B^{10} divides into its major terminal bronchi.

TABLE X. THE POSTERIOR BASAL BRONCHUS (B^{10})

	PITEL AND BOYDEN (50 SPEC.)		BERG AND BOYDEN (60 SPEC.)		TOTAL (110 SPEC.)	
I. Origin of B^{10}						
1. B^{10} arises alone		6%		6.7%		6.4%
2. B^{10} arises in conjunction with B^{9}		90%		93.3%		91.8%
1) From a trunk dividing into B^{9} and B^{10}	56%		66.7%		61.8%	
2) Collaterally with B^{9} and B^{7+8}	30%		13.3%		20.9%	
3) Collaterally with B^{8} and B^{9}	4%		10.0%		7.3%	
4) Collaterally with $B^{8}b$	0%		1.7%		0.9%	
5) Collaterally with B^{7} and B^{9}	0%		1.6%		0.9%	
3. B^{10} arises collaterally with B^{7+8} and BX^{*} *(9)*		4%		0.0%		1.8%
II. Terminal rami						
1. B^{10} divides into $B^{10}a$ and $B^{10}b$	96%		100%		98.2%	
2. $B^{10}a$ displaced to B^{9}	4%		0%		1.8%	
III. *Collateral branches* above terminal rami						
1. One to three dorsal rami, BX^{*} *(10)*		84%		85%		84.6%
2. A high paravertebral branch		36%*		30%		32.8%
1) Having common origin with BX^{*} *(10)*	24%					
2) Having separate origin	12%					

*In the 50 specimens of Pitel and Boyden, this 36 per cent of paravertebral rami occurred in specimens already having one or more subsuperiors, BX^{*} *(10)*. Therefore the main stem of B^{10} was free of large collateral branches in only 16 per cent.

The arterial pattern: In discussing the mode of division of the basal portion of the left pulmonary artery, reference has already been made to the fact that the basal artery bifurcates unexpectedly into A^{10} and A^{7-9} in 46 per cent of fifty specimens (cf. Fig. 2, *C; B,* Plate 4). In another 42 per cent, it divides into an expected A^{7+8} and A^{9+10}. In the remaining 12 per cent, it is either one stem of a trifurcation (Plate 4, #7) or associated irregularly with A^{7} (8 per cent of fifty specimens).

TABLE XI. DISTRIBUTION OF BX^{*} *(10)*

	PITEL AND BOYDEN (50 SPEC.)	BERG AND BOYDEN (60 SPEC.)	TOTAL (110 SPEC.)
I. Sector			
1. Wholly posterior	12%	36.7%	25.5%
2. Posterior and posterolateral	38%	25.0%	30.9%
3. Posterolateral only	22%	15.0%	18.2%
4. Posterolateral and lateral	6%	1.7%	3.6%
5. Paravertebral and posterior	2%	5.0%	3.6%
6. Paravertebral, posterior, and posterolateral	4%	1.7%	2.7%
	84%	85 %	84.5%
II. Angle made with anterior surface of lobe			
1. 75-100 degrees (posterior)	72%	62%	66.3%
2. 45-50 degrees (posterolateral)	12%	23%	18.2%
	84%	85%	84.5%*

*Note that the posterolateral sector of the subsuperior zone is supplied by BX^{*} *(10)* in nearly two-thirds of the specimen having a subsuperior of this type. (Add items 2, 3, 4, and 6.)

The venous pattern: It will be recalled that the posterior basal segment is separated from the lateral basal by V^9 (Plate 1). Otherwise the principal drainage of the posterior basal segment is mediated by V^{10}. This has two principal tributaries—a segmental vein ($V^{10}a$) and an intersegmental vein ($V^{10}b$). In 92 per cent of fifty specimens, V^{10} empties into the inferior basal vein (Plate 1). In the remaining 8 per cent it is split into two veins, one draining into the inferior basal, the other into the superior basal vein (Table I).

6. *The Subsuperior Zone.—*

The bronchial supply: This zone receives its name from a dorsal bronchus (B^*)—the second dorsal of Aeby—which arises either from the basal trunk of the lobe (9 per cent of 110 specimens) or from the common stem of B^{9+10} (18 per cent). This bronchus differs from the corresponding bronchus of the right lower lobe in two major respects: first, in its less frequent occurrence—27 per cent as against 61 per cent—and, second, in its distribution—that of the left (Table XII) supplying the posterolateral and often the lateral (as well as the posterior) sector of the zone which is interpolated between superior and basal segments, that of the right supplying primarily the posterior sector. For these reasons, Berg and Boyden concluded that any dorsal bronchus which supplied the territory of the left B^* should be considered as an *accessory subsuperior bronchus* (BX^*)—namely the high dorsal branch of B^9 [BX^* *(9)*] and the high dorsal branch of B^{10} [BX^* *(10)*]. Indeed, one or both of these (third dorsal level of Aeby) supply the subsuperior zone exclusively in 72.7 per cent of 110 specimens (Table XII). Their occurrence and distribution are summarized in Tables IX, XI, XII, XIII, and XIV, and their origin on the bronchial tree is illustrated in Fig. 1.

TABLE XII. POSITION AND ORIENTATION OF SUBSUPERIORS PROPER (B^*)

	PITEL AND BOYDEN (50 SPEC.)	BERG AND BOYDEN (60 SPEC.)	TOTAL (110 SPEC.)
I. Level on bronchial tree			
1. Above origin of B^{7+8}	14%	5%	9.1%
2. From common stem of B^{9+10}	12%	24%	18.2%
	26%	29%	27.3%
II. Position of orifice on circumference			
1. Posterior (or slightly posterolateral)	14%	12%	12.7%
2. Posterolateral	6%	12%	9.1%
3. Lateral (or slightly posterolateral)	2%	5%	3.6%
4. Medial	4%	0%	1.8%
III. Angle of subsuperior bronchus with anterior surface			
1. 40-45 degrees (i.e., posterolateral)	14%	23%	18.2%
2. 20-30 degrees (i.e., almost lateral)	8%	3%	5.4%
3. 80-100 degrees (i.e., posterior)	4%	3%	3.6%
IV. Distribution on surface			
1. Posterolateral and lateral	20%	14%	16.4%
2. Posterior, posterolateral, and lateral	0%	10%	5.4%
3. Posterolateral only	2%	3%	2.7%
4. Posterior and posterolateral	0%	2%	0.9%
5. Paravertebral only	4%	0%	1.8%
			27.2%*

*Note that the posterolateral sector of the subsuperior zone is supplied by B^* in 94% of the specimens having a subsuperior bronchus. (Add items 1, 2, 3, and 4.)

TABLE XIII. OCCURRENCE OF SUBSUPERIOR BRONCHI (*B** AND *BX**)

	PITEL AND BOYDEN (50 SPEC.)	BERG AND BOYDEN (60 SPEC.)	TOTAL (110 SPEC.)
1. Frequency of occurrence according to levels			
*B** only (level d_2)*	4%	6.6%	5.5%
*BX** only (level d_3)†	74%	71.7%	72.7%
*B** plus *BX** (both levels)	22%	21.7%	21.8%
2. Number of subsuperiors per specimen			
One subsuperior	18%	21.7%	20.0%
Two subsuperiors	32%	61.7%	48.2%
Three subsuperiors	26%	16.6%	20.9%
Four subsuperiors	24%	0.0%	10.9%
3. Frequency of occurrence of various types of subsuperiors			
*B**	26%	28.3%	27.3%
BX (9)*	80%	66.7%	72.7%
BX (10)*	84%	85.0%	84.5%

*Supsuperiors proper, arising below B^8 and above the keel which separates B^9 and B^{10}.

†Accessory subsuperiors, arising below the keel which separates B^9 and B^{10}: either from the upper portion of the trunk of B^{10} [*BX* (10)*] or the upper portion of the trunk of B^9 [*BX* (9)*], or from both.

Mention has been made of the frequent extension of the subsuperior bronchi into the anterolateral sector of the zone. In other words, ***B****, ***BX* (9)***, and occasionally ***BX* (10)*** invade the territory of the lateral ramus of the anterior basal bronchus (*B^8a*). In our fifty specimens (footnote, Table VII), *B^8a* was supplemented or replaced by *X^8a* branches of the subsuperior bronchi

TABLE XIV. DETAILED ANALYSIS OF NUMBERS OF SUBSUPERIORS PER SPECIMEN

	PITEL AND BOYDEN (50 SPEC.)	BERG AND BOYDEN (60 SPEC.)	TOTAL (110 SPEC.)
1. *One subsuperior*			
Level d_2 (*B**)	4%	6.7%	5.45%
Level d_3			
BX (9)*	12%	6.7%	9.1%
BX (10)*	2%	8.3%	5.45%
	18%	21.7%	20.00%
2. *Two subsuperiors*			
Both levels			
*B** and *BX* (9)*	0%	1.7%	0.9%
*B** and *BX* (10)*	4%	11.7%	8.2%
Level d_3			
BX (9)* and *BX* (10)*	26%	43.3%	35.5%
Two *BX* (10)*	2%	5.0%	3.6%
	32%	61.7%	48.2%
3. *Three subsuperiors*			
Both levels			
Two *B**, one *BX* (10)*	2%	1.6%	1.8%
One *B**, one *BX* (9)*, one *BX* (10)*	8%	6.7%	7.3%
One *BX* (9)*, two *BX* (10)*	12%	8.3%	10.0%
Three *BX* (10)*	4%	0 %	1.8%
	26%	16.6%	20.9%
4. *Four subsuperiors*			
Both levels			
One *B**, three *BX* (10)*	2%	0%	0.9%
One *B**, one *BX* (9)*, two *BX* (10)*	6%	0%	2.7%
Level d_3			
One *BX* (9)*, three *BX* (10)*	16%	0%	7.3%
	24%	0%	10.9%

in one-third of the lobes (35.4 per cent). Such variations are illustrated in Plates 4, *B*, and 5.

Origin of the subsuperior arteries: The arteries that accompany the subsuperior proper *(A*)* arise at different levels on the trunk and branches of the inferior pulmonary artery. In 4 per cent of fifty lobes they arise from the basal artery, in 6 per cent from the common trunk of A^{7-9}, in 8 per cent from the common trunk of A^{9+10} (Plate 3), in 6 per cent from A^{10}, and in one instance (2 per cent) from A^{9}.

The arteries accompanying *BX* (9)* arise prevailingly from A^{9} (56 per cent: Plate 4, *B*), next in frequency from $A^{10}a$ (10 per cent), then less frequently from the trunks of A^{7-9} or A^{7+9} (8 per cent), A^{9+10} or $A^{9+10}a$ (6 per cent), and $A^{8}a$ (4 per cent). In two specimens there were two arteries to a bronchus—from adjacent sides of A^{9} and A^{10}.

The arteries accompanying *BX* (10)* arise prevailingly from A^{10} (42 per cent: Plate 4, *B*), then with about equal frequency from A^{9} (14 per cent), $A^{10}a$ (12 per cent), $A^{10}b$ (12 per cent), and infrequently from the trunks of A^{9+10} (4 per cent) or A^{7-9} (2 per cent). In one specimen, there were two arteries to one bronchus, from adjacent sides of A^{9} and A^{10}.

The veins of the subsuperior zone: Since B^{*} is directed primarily to a posterior lateral sector of the zone (Plate 1) it is to be expected that V^{*}, the vein lying on the clockwise side of B^{*}, should pass between B^{7+8} and B^{9} to gain the superior basal vein or one of its tributaries *($V^{8}a$)*. Such is the case in ten of the thirteen specimens in which it occurs (cf. Plate 2, right figure). In the remaining three specimens, V^{*} drains into V^{6} (see $VX^{8}a$ branch of V^{*}, Plate 5) or into the common basal vein (Plate 3).

A similar course is followed by *VX* (9)*—the vein which lies on the clockwise side of *BX* (9)*. In thirty-two of the thirty-seven specimens in which it occurs, *VX* (9)* runs between B^{8} and B^{9} to enter $V^{8}a$ (cf. Plate 2, right figure). In the remaining five specimens, it courses between B^{9} and B^{10} to enter V^{9}. In three of the five, however, it first joins V^{*} or *VX* (10)* before entering V^{9}.

The third subsuperior vein (Plate 1) is *VX* (10)*. Since in one-half the specimens there are two or more subsuperior bronchi of this type (Table XIV), we shall restrict the description to the vein accompanying the highest *VX* (10)*, if more than one is present, for the lower ones are less significant and drain into V^{10}. The principal *VX* (10)* lies on the clockwise side of *BX* (10)*. Commonly, it passes through the crotch between B^{9} and B^{10} (Plate 1). There, in about one-half the thirty-four specimens in which it occurs, it empties directly into V^{9}, the most centrally placed of the basal veins. In most of the remaining specimens it joins some other centrally placed vein—even forming one of its own in conjunction with *VX* (9)* or V^{*} (Plate 4, *A* and *B*). In five cases, however, it is displaced laterally to enter $V^{8}a$ between B^{8} and B^{9}.

SUMMARY OF STRIKING DIFFERENCES BETWEEN LEFT AND RIGHT LOWER LOBES

The key to the differences of pattern in left and right lower lobes is to be found in the position of the medial basal bronchus *(B^{7})*. In the right lung it

is medially placed and therefore distributes to both anterior and paravertebral surfaces. Because of this, the bronchus embraces the inferior pulmonary vein in one-fourth of the lobes. In another one-fifth its two rami arise separately—one on either side of the vein (Ferry and Boyden, 1951). In the left lung the medial basal bronchus arises in common with B^8, the lateral basal bronchus. Accordingly, B^7 never distributes to the paravertebral surface but always remains on the lateral side of the pulmonary ligament. This holds true even in the 7 per cent of specimens in which B^7 arises independently from the anteromedial portion of the basal stem. Correspondingly it never "tangles" with the inferior pulmonary vein. Indeed, the only occasion in which the left pulmonary vein and its large branches pass superficial to B^7 is when the latter arises anomalously from B^{10} (Fig. 3).

Because of this fundamental difference in the two sides, the mode of entrance into the two lobes (for purposes of exposing the large veins and deeper bronchi) is quite different. On the right side a cleavage plane marked by V^8b is entered and the medial basal segment is reflected medially. On the left, one proceeds through the pulmonary ligament to an underlying cleavage plane formed by V^7b, and then reflects the medial basal segment laterally.

Thus exposed, the pattern of the great veins is found to be essentially similar—namely, the inferior pulmonary is formed by the confluence of a superior vein (V^6) and a common basal vein; the latter in turn is formed by the union of superior and inferior basal veins. However, the composition of the basal veins differs somewhat in the two lobes. On the left side, V^9 commonly enters the inferior basal vein (Plate 1) instead of the superior basal, as on the right side. Furthermore the left inferior pulmonary is much less variable. In 14 per cent, the right pulmonary is formed by union of three or four major veins receiving substantial tributaries from the middle lobe and even from the right upper lobe (Table III, Ferry and Boyden). Such an arrangement has never been encountered by us in the left lower lobe.

In the left superior segment, due to the greater development of the medial ramus (B^6a) the superior vein usually receives three major tributaries (V^6a, b, and c) instead of the two on the right (V^6b and c). Correspondingly, multiple arteries to the left superior segment are more common, occurring in 36 per cent of specimens, as against 20 per cent for the right superior segment. Furthermore, they arise from the interlobar portion of the pulmonary artery *above* the level of the lingular arteries—a point of great importance in lobectomy—whereas on the right, they occur both above and below the middle lobe arteries. Curiously enough, the left superior segmental bronchus (B^6) is "conservative" in its origin. Only one instance (in 110 specimens) was found in which it arose as two separate stems (Plate 4, #7). In this case the lower stem (B^6a) arose from the medial side of the basal trunk. In the right lung, two separate origins of B^6 were encountered in 6 per cent of 100 specimens. In these cases the lower stem tends to originate on the lateral side and to involve ramus B^6c.

The subsuperior bronchi (B^*) are fewer in number on the left side—27 per cent as against 61 per cent on the right. Furthermore, they are oriented in

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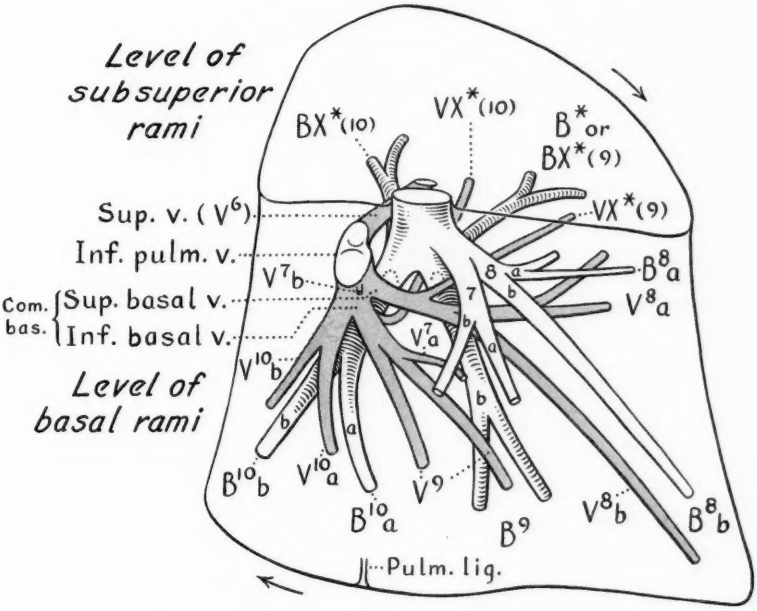

Scheme showing common arrangement of veins in basal segments

(Inf. pulm. v. retracted medially)

Plate 1.—Diagram of truncated left lower lobe. The superior segment has been removed to show subsuperior rami.

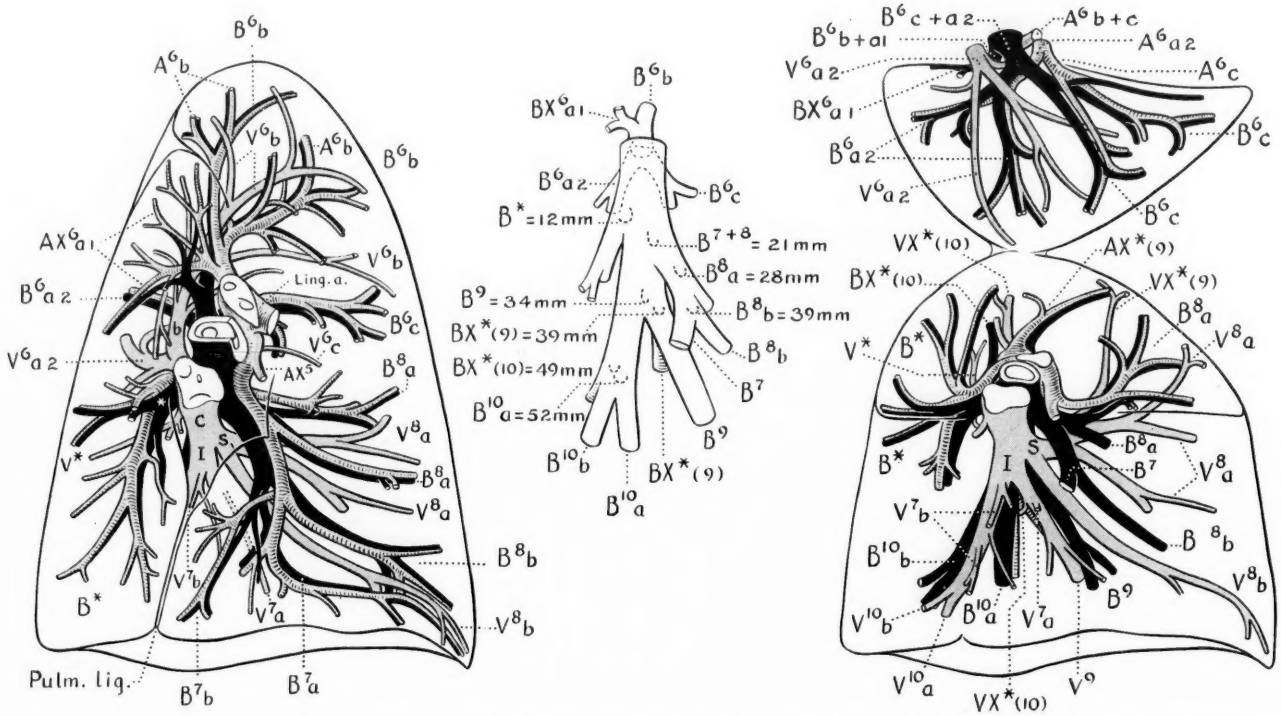

Plate 2.—Specimen #44. *Left figures*, dissected anterior surface; *middle figure*, bronchial tree; *right figure*, deep dissection with superior segment tipped back on a hinge. Bronchi in black, arteries in red, veins in blue color. *C*, common basal; *S*, superior basal; *I*, inferior basal vein.

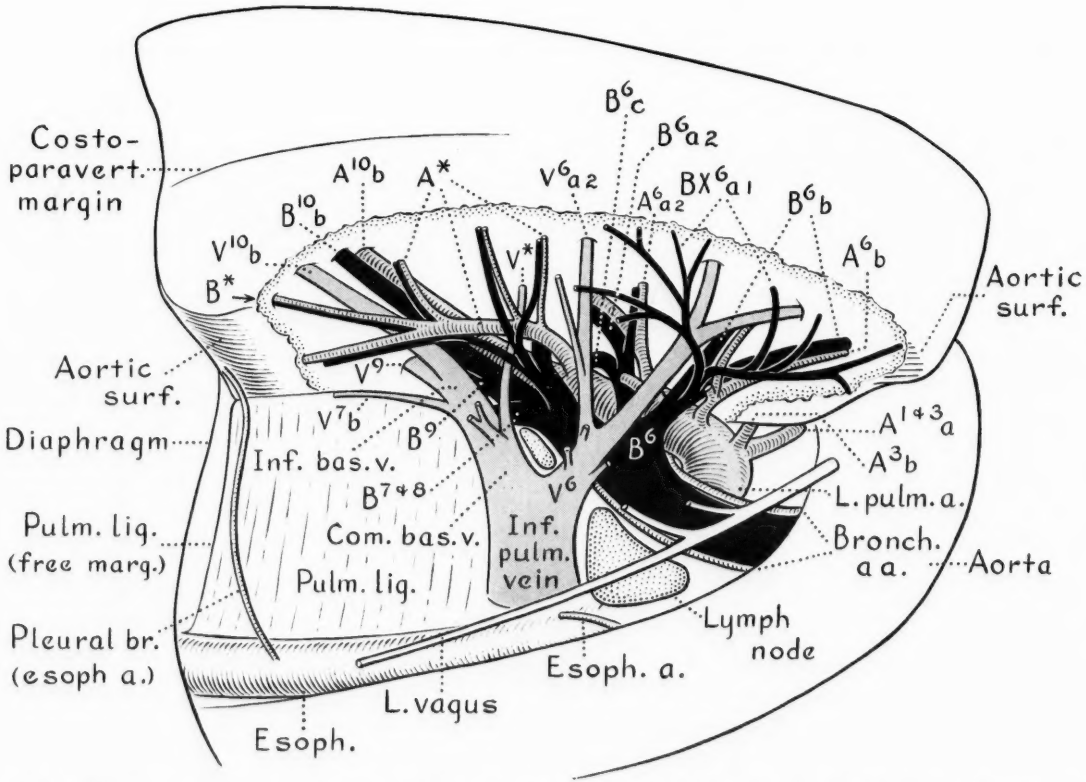

Plate 3.—Specimen #44. Left lower lobe elevated and drawn in situ. Small veins on stem of V^6 and common basal vein are bronchial veins.

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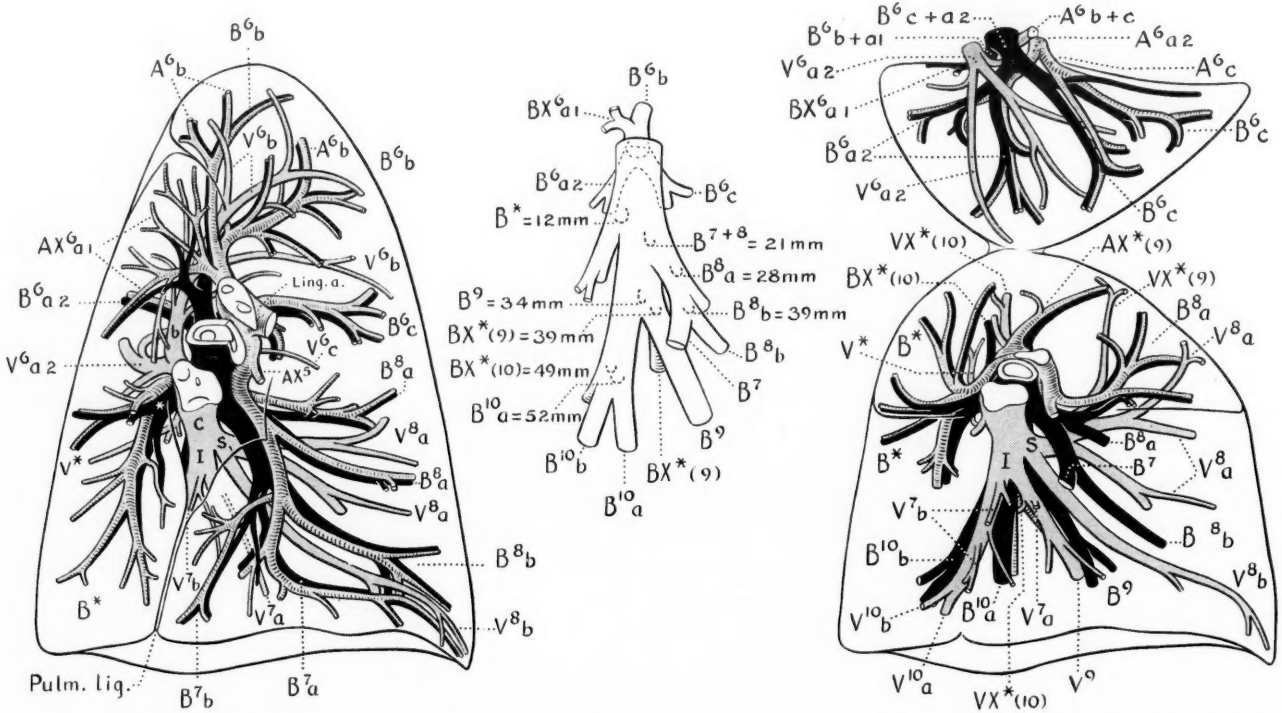

Plate 2.—Specimen #44. *Left figures*, dissected anterior surface; *middle figure*, bronchial tree; *right figure*, deep dissection with superior segment tipped back on a hinge. Bronchi in black, arteries in red, veins in blue color. *C*, common basal; *S*, superior basal; *I*, inferior basal vein.

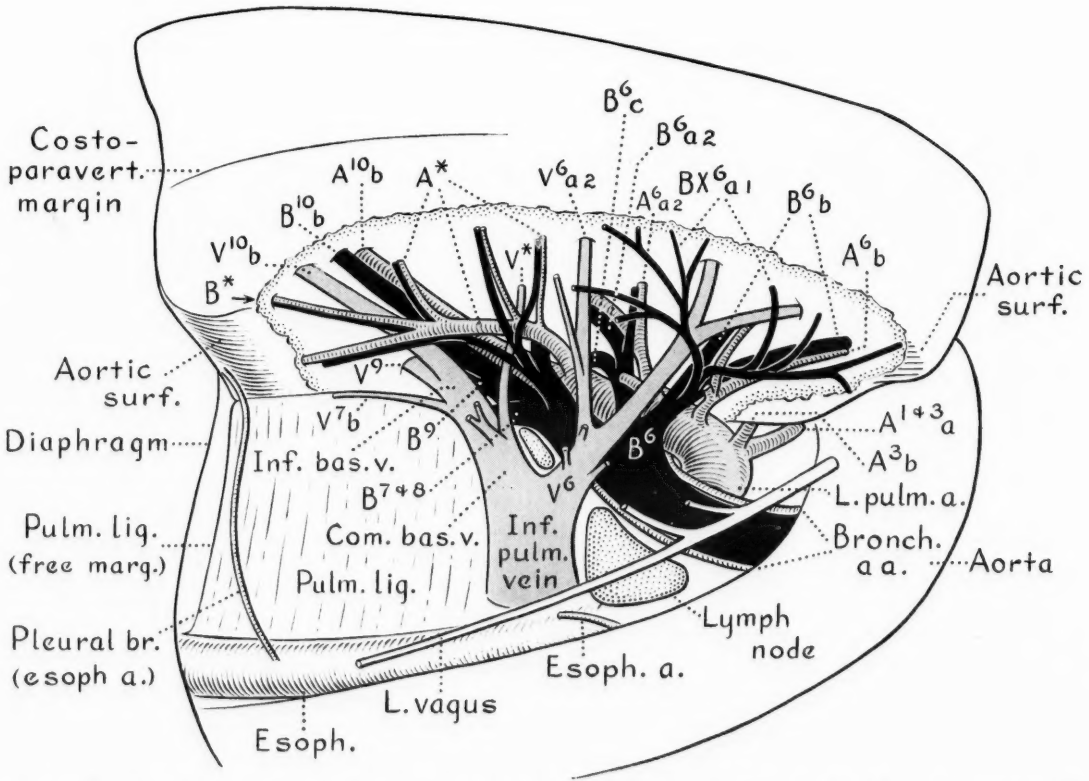

Plate 3.—Specimen #44. Left lower lobe elevated and drawn in situ. Small veins on stem of V^6 and common basal vein are bronchial veins.

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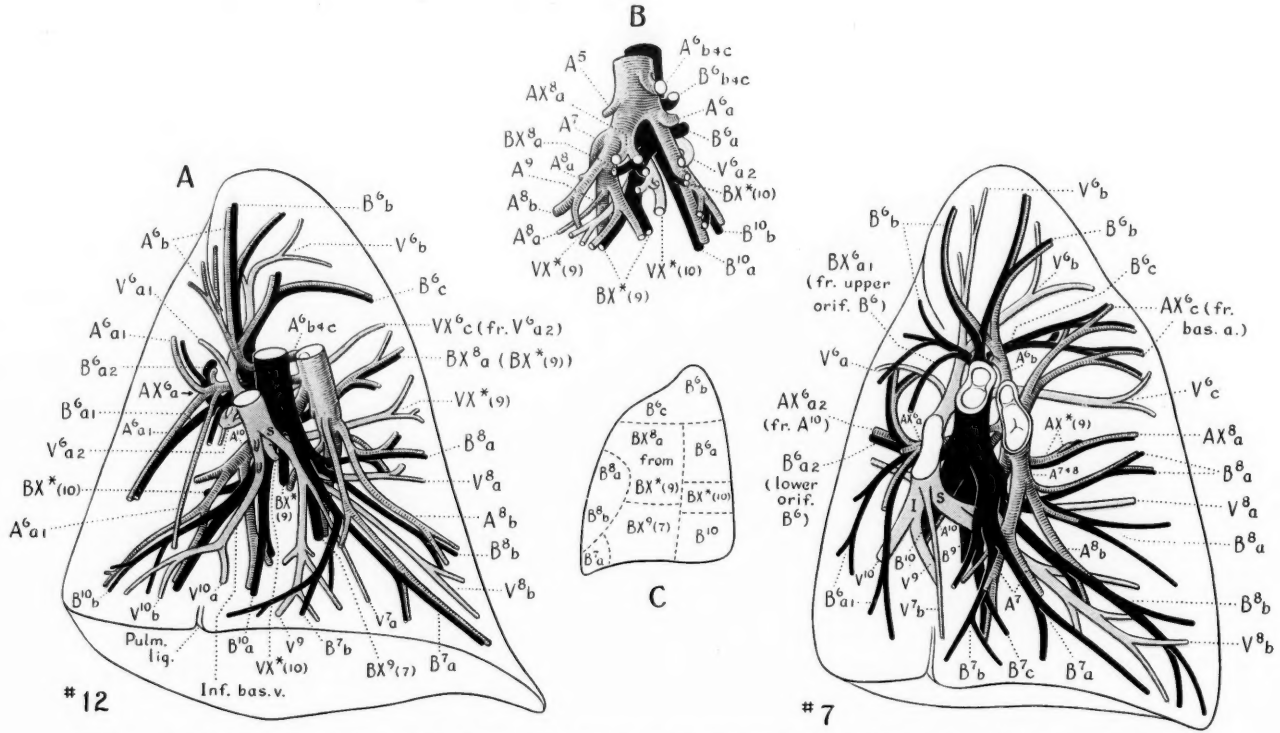

Plate 4.—*A*, *B*, and *C*, three views of Specimen #12. Specimen #7 illustrates double origin of bronchi of superior segment.

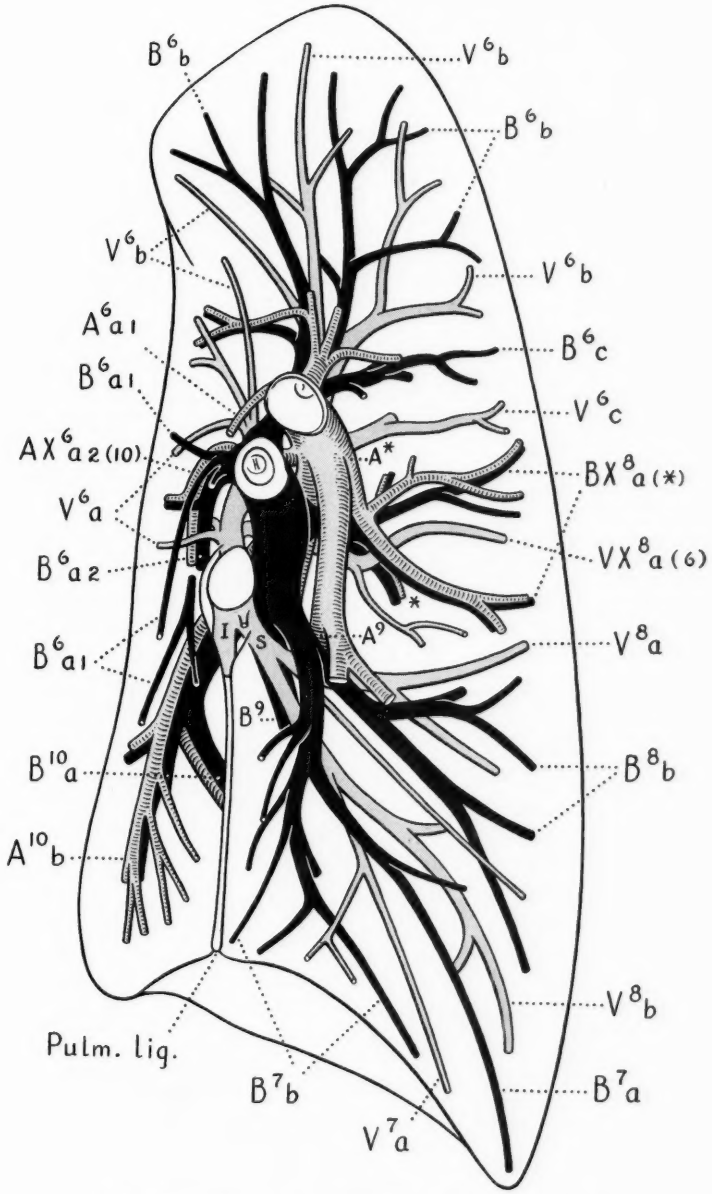

Plate 5.—Specimen #46. Dissection of anterior surface.

a posterolateral instead of a posterior direction. Therefore, in the absence of B^* on the left, the posterior lateral portion of the zone is supplied by the highest branch of B^9 (the B^9a ramus of the right side)—namely *BX* (9)*. One of the most characteristic features of the left subsuperior zone is its invasion of the anterior surface of the anterior basal segment. This occurs in over one-third of the lobes.

Turning to the basal segments, the anterior basal is the most variable of the left basal bronchi, whereas on the right it is the most constant. One or the other of its two rami is missing in 17 per cent of 110 specimens; B^8a is supplemented or wholly replaced by subsuperior bronchi in 35 per cent; B^8b fails to reach the diaphragmatic surface in 19 per cent; finally, the common stem of B^7 and B^8 divides unevenly into B^8a and $B^{7+8}b$ in 20 per cent. In addition, B^9 (the lateral basal bronchus) is missing on the left side in 11 per cent of 110 specimens, being usually displaced onto some portion of the B^{7+8} stem. As a result of these related variations there is a profound change in the arterial pattern of the left side. Whereas in the right lung the basal portion of the inferior pulmonary artery divides into A^9 and A^{10} in nearly one-half of the lobes (44 per cent), the left basal artery divides unevenly into A^{10} and A^{7-9} in 46 per cent of specimens. In consequence of this unexpected grouping of segmental arteries, A^9 arises anomalously from the deep side of A^7a, A^8, A^8a, or A^8b in 22 per cent of lobes. In addition, the pattern of the anteromedial basal arteries conforms to the bronchial pattern in less than one-third of the specimens. This constitutes a considerable hazard to the individual resections of the first three basal segments.

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Announcement

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Notice of Annual Meeting

The 34th annual meeting of the American Association for Thoracic Surgery will be held May 3, 4, and 5, 1954, in Montreal, Que., Canada. Headquarters will be at the Sheraton-Mount Royal Hotel.

Requests for Hotel Reservations

These should be mailed *directly* to the Reservation Manager, Sheraton-Mount Royal Hotel, Montreal, Que., Canada. Please mention this Association, type of accommodation desired, date, and approximate hour of arrival. If accommodations are desired elsewhere, please communicate directly with hotel of your choice.

Thoracic Surgical Forum

After due consideration, the Council has decided to try an experiment by assigning one morning session to a "Thoracic Surgical Forum" for the presentation of short papers on current thoracic research, anatomy, modified surgical techniques, etc. Papers at this session will be limited sharply to ten minutes (including the showing of lantern slides or other illustrative material) with three minutes as the maximum discussion of each paper.

These papers will be published together in one issue of THE JOURNAL OF THORACIC SURGERY. Maximum length: 3,000 words without illustrations. If illustrated, an appropriate number of words must be subtracted for each cut used.

Abstracts for Papers

Abstracts for the presentation of papers at the 1954 meeting, including papers before the Forum, must be received on or before January 1, 1954, otherwise they will not be considered by the Program Committee.

Abstracts should be labeled "For Forum" or "For Regular Program."

Five, repeat five, copies of each abstract should be sent to the Secretary of the Association, Dr. Paul C. Samson, 2938 McClure St., Oakland 9, Calif. They should not be sent to the Editor.

The abstracts should contain from 200 to 250 words and accurately reflect the contents of the completed paper.

Applications for Membership

Applications for Associate Membership in the Association must be received by the Membership Committee not later than Jan. 1, 1954, to be considered for action in Montreal. Applications received beyond this deadline will be deferred for consideration until 1955.

Applicants must be sponsored by three Active or Senior Members of the Association. In addition to the sponsoring statement on the application form, the sponsors *must* submit a separate letter concerning the applicant direct to Dr. Lyman H. Brewer, III, Chairman of the Membership Committee, 2010 Wilshire Blvd., Los Angeles, Calif. Completed application forms should likewise be sent to Dr. Brewer. These regulations are at the direction of the Council and were made effective in 1952 at Dallas.

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